ABILITY OF POSTPARTUM CURETTAGE TO ACCELERATE MATERNAL RECOVERY IN SEVERE PRE ECLAMPSIA

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BONAFIDE CERTIFICATE

This is to certify that this dissertation is the bonafide work of **Dr.K.Karthika** on **"ABILITY OF POSTPARTUM CURETTAGE TO ACCELERATE MATERNAL RECOVERY IN SEVERE PRE ECLAMPSIA"** during her M.S.,(Obstetrics and Gynaecology) course from April 2011 to April 2014 at the Government Stanley medical college and Raja Sir Ramasamy Mudaliar Lying-in Hospital, Chennai.

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DECLARATION

I, **Dr.K.Karthika**, solemnly declare that the dissertation titled **"ABILITY OF POSTPARTUM CURETTAGE TO ACCELERATE MATERNAL RECOVERY IN SEVERE PRE ECLAMPSIA"** is done by me at RSRM Lying in Hospital ,Stanley Medical College and Hospital under the guidance of **Prof. Dr.VASANTHAMANIM.D;D.G.O.** Professor of Obstetrics and Gynaecology, Stanley Medical College& RSRM Lying in Hospital, Chennai 13.

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CONTENTS

INTRODUCTION

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Hypertensive disorders during pregnancy continue to be a major cause of maternal and perinatal morbidity and mortality worldwide. In developing countries they are second only to anemia with approximately 3- 10 %of all pregnancies.

Pritchard et al observed “in order to effect a complete cure from preeclampsia the chorionic villi must be expelled or surgically removed .Rodger et al and Music et al postulated that the endothelial cells are affected by a cytotoxic factor produced by the trophoblastic cells which is responsible for the pathophysiology of preeclampsia[1].

One such theory states that “the decidua and amniotic fluid contains a toxin called HYSTEROTONIN which acts as a pressor substance leading to the development of preeclampsia. It is found in women with preeclampsia. Resolution of preeclampsia and eclampsia occurs by delivery and complete removal of trophoblastic cells.

Preeclampsia resolves only after all gestational products particularly the placenta and decidua have been removed or have ceased to function. Therefore removal is the best means of cure of the disease.

We consider the above theory and therefore by immediate postpartum curettage we try to remove all the decidual tissues therefore hastening the recovery. Hence in patients with severe preeclampsia accelerated recovery following delivery will minimize serious and life threatening maternal complications thereby minimizing the patients stay in intensive care and prolonged hospitalisation.

This thesis is done to study the effect of immediate postpartum curettage on the resolution of clinical and laboratory indices associated with severe preeclampsia.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

PREECLAMPSIA

Preeclampsia is a pregnancy-specific disorder defined clinically as hypertension and proteinuria occurring after 20 weeks gestation. Preeclampsia is a common complication occurring in the second trimester and is potentially dangerous to both mother and fetus. Preeclampsia occurs in 3-10% of pregnancies and approximately 50 000 women worldwide die from this disease every year[2].

The pathogenesis of preeclampsia is not fully understood. Considerable research over more than five decades has led to the notion of preeclampsia as a multifactorial syndrome with maternal and fetal interaction.

DEFINITIONS

According to the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy[3], the following classifications are recommended.

Preeclampsia:

A syndrome defined by hypertension and proteinuria after 20 weeks of gestation. Hypertension is defined as a systolic blood pressure level of 140mm/Hg or higher or a diastolic blood pressure level of 90 mm /Hg or higher occurring after 20 weeks gestation in a woman with previously normal blood pressure. Proteinuria is defined as urinary excretion of 0.3 gram of protein or higher in a 24-hour urine specimen (or protein dipstick reading equal to or higher than 1+ on more than one midstream urine sample six hours apart).

Eclampsia:

The occurrence of seizures in a preeclamptic woman, where the seizures cannot be attributed to other causes.

Gestational hypertension:

Blood pressure elevation without proteinuria developing in a woman after 20 weeks' gestation, with blood pressure levels returning to normal postpartum.

Chronic hypertension:

Hypertension that is observable before pregnancy or before 20weeks' gestation. Hypertension that is primarily diagnosed during pregnancy and which persists beyond the 42nd day postpartum is also classified as chronic hypertension.

Superimposed preeclampsia:

Preeclampsia developing in a patient with chronic hypertension.

PATHOGENESIS

PREECLAMPSIA AND DEFECTIVE PLACENTATION

The complex molecular inheritance between the mature blastocyst and the uterine endometrium which is primed hormonally is the final result of a successful implantation[4]. The function of the uteroplacental unit is to secure an adequate exchange of nutrients, gases and metabolic end products. These functions are mediated by increased blood flow through the uterus. During the early phase of placental development, extravillous cytotrophoblasts (stemming from the outer cell-layer of the blastocyst) stream out of the anchoring villi, penetrating the syncytiotrophoblast layer, entering the decidua and invading and

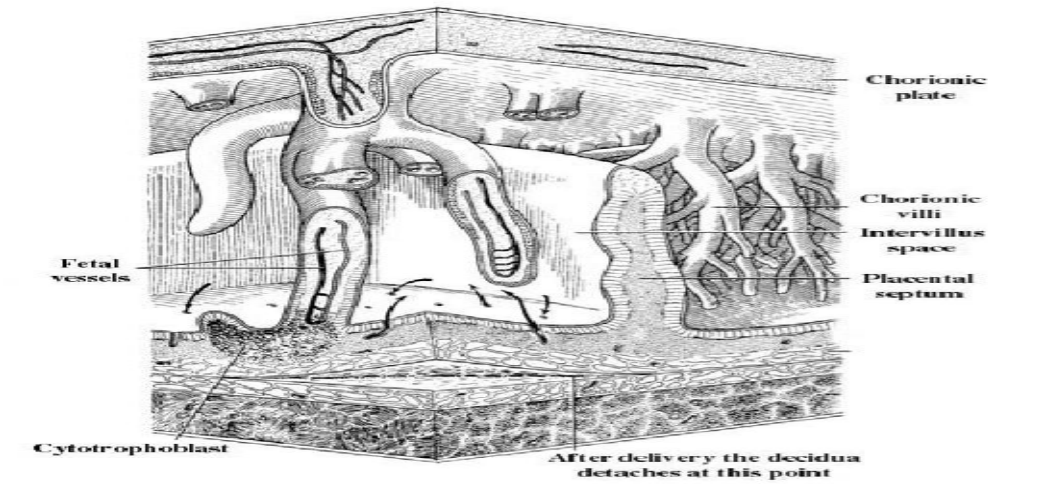
transforming the maternal spiral arteries. The major structural alterations occurring in these spiral arteries in early pregnancy (so-called "physiological changes") normally transform the arteries to vascular channels outside maternal vascular control, allowing an increased maternal blood supply to the placenta [5] in the last part of the pregnancy (figure I).

Trophoblast invasion of the decidua and maternal myometrium is thought to be a key event in early placentation[6]. Reduced trophoblast invasion may represent one of the basic defects leading to preeclampsia[7].This reduced invasion of extracellular trophoblast is followed by a shallow transformation of the spiral arteries [6], leaving them narrow, tortuous and thickwalled. Besides remaining non-transformed, the spiral arteries in preeclampsia can have areas of lipid deposition in the vessel wall, a phenomenon known as acute atherosis. The name was given to the changes, first observed in 1945, as a result of the morphological resemblance of early phases of atherosclerotic lesions in vessels[8]. There is still some controversy and insufficient knowledge regarding the etiology and molecular mechanisms behind the formation of acute atherosis. This is partly due to the difficulty in obtaining

sufficient and adequate tissue from the placental bed, as well as a lack of relevant animal models.

Normal placental function relies on adequate maternal blood supply through the wholly or partially transformed spiral arteries in the placental bed (figure I). Any process that results in mural or occluding thrombi in these arteries reduces the flow and can lead to hypoxia and ischemia as well as infarcts in the placental tissue, i.e. reduced placental function, which is more common in preeclampsia than in uncomplicated pregnancies [9].

Term Placental Structure



The arrows represent blood flow from the decidual arteries into the intervillous spaces and back into the venous blood (modified after Junqueira LC et al.Basic Histology. Appleton and Lange 1989: 452-458)

Oxidative stress and maternal endothelial dysfunction is due to the toxic compounds released by the hypoxic placenta into the maternal circulation[10,11]. Acute atherosis in spiral arteries is associated with an increased risk of thrombosis and thereby placental ischemia and infarctions [9].Bearing in mind the multifactorial aspects of preeclampsia, defect in placentation additionally seems to be related to immunological processes such as decreased trophoblast expression of human leucocyte antigen-G (HLA-G) [12], as well as interaction with maternal lymphocytes such as uterine natural killer (NK) cells [13]

INFLAMMATION AND ENDOTHELIAL DYSFUNCTION

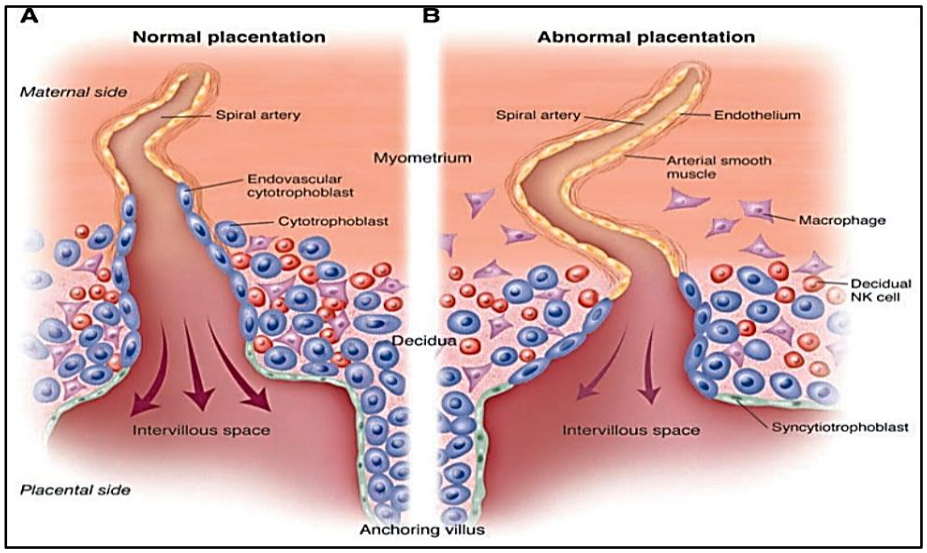
Endothelial cells represent a semipermeable barrier between the vessel wall and the blood flow in the vascular system [14]. Hemostasis is mainly regulated by the endothelial lining of the cells via a series of receptors for proteins, hormones, lipid transports as well as through cell-cell interactions [15].

"Endothelial dysfunction" has not been precisely defined, but the term is used to indicate change in endothelial properties with activation and abnormal function [16]. Evidence suggests that endothelial activation and low grade maternal inflammation is present in all pregnancies, there

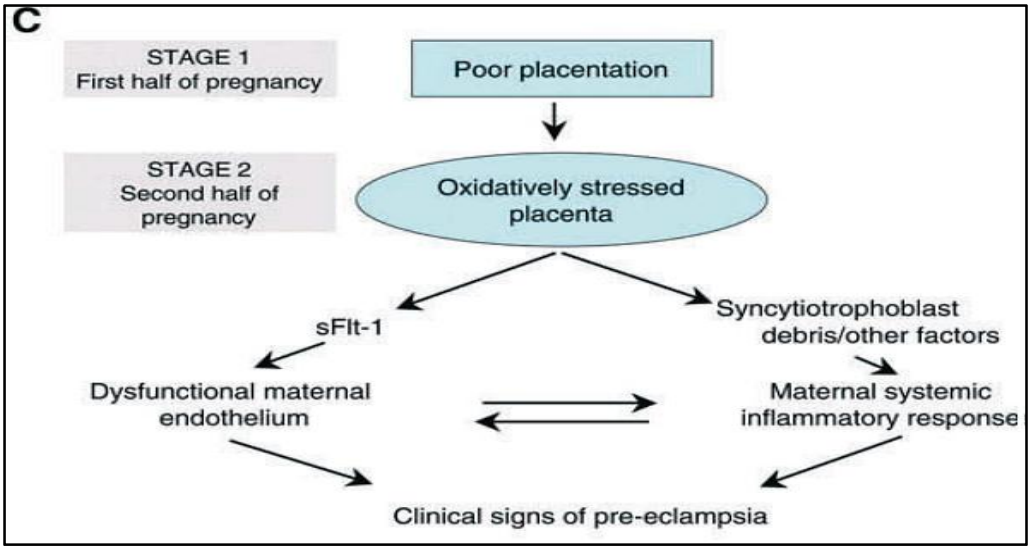
being merely a different gradient in preeclampsia, with a stronger inflammatory response [17]. The central pathological features of the maternal syndrome may thus include the presence of an excessive maternal inflammatory response, resulting in dysfunction, activation and peripheral vasoconstriction with reduced circulating volume, which in turn leads to oxidative stress [17].

PREECLAMPSIA AND THE TWO-STAGE-MODEL

The present concept of the pathogenesis of preeclampsia involves a two-stage model. Stage 1 refers to a defect and reduced trophoblast invasion into the maternal spiral arteries (with reduced physiological arterial changes or remodeling) leading to reduced placental perfusion. Stage 2 refers to a generalized dysfunction and activation of the endothelium and finally development of the maternal syndrome[18,19].



Several factors have been proposed as the link between the two stages of preeclampsia such as the angiogenic factors[20-22], oxidized lipids[23] and syncytiotrophoblastic debris[24] which are released by the placenta into the maternal circulation. Recent theories state that the angiogenic associated factors like sFlt (soluble fms like tyrosine) and endoglin released from a hypoxic placenta are responsible for the endothelial damage[25].



Oxidative stress in preeclampsia

The biochemical imbalance between antioxidant protection and free radical damage is defined as oxidative stress[26]. It arises from excessive generation of free radicals and inadequate endogenous antioxidant capacity [27,28]. Reactive oxygen species (ROS) are constantly

produced as by-products of normal oxidative metabolism in mitochondria and other cellular reactions [29,30]. All organisms possess a range of enzymatic and non enzymatic antioxidant systems, which serve to protect against the harmful oxidative reactions that occur as a consequence of this endogenous ROS production. Some of the enzymatic antioxidents are glutathione reductase, superoxide dismutase and catalase. Some of the non enzymatic antioxidents are vitamins A,C& E , flavonoids and glutathione.[31]Under certain conditions, an increase in oxidants and a decrease in antioxidants cannot be prevented, and the oxidant/ antioxidant balance shifts towards the oxidative state. This oxidative stress is responsible for the pathological changes and endothelial dysfunction in preeclampsia [32-34].

Pregnancy itself is a condition of increased oxidative stress [35], and may contribute to endothelial dysfunction and the clinical development of preeclampsia [36,37]. There is considerable evidence supporting an increase in products of oxidative stress in women with preeclampsia, demonstrated both in the placenta [21,38] and in maternal peripheral blood cells [33], as well as in the maternal circulation [39]. There is still some controversy with respect to the results for latter compartment [40,41].

A number of circulating compounds may under certain circumstances enhance oxidative stress. They include lipoproteins, lipids, proteins, glucose and advanced glycation end products (AGEs).An established method for measuring the end products of increased oxidative stress is to measure the degree of lipid peroxidation. Lipid peroxidation occurs when ROS interact with polyunsaturated fatty acids in membranes or lipoproteins [42]. In preeclampsia, there is development of excessive maternal hyperlipidemia, and especially hypertriglyceridemia, as compared to normal pregnancy [43,44] and these changes are present long before the onset of preeclampsia [45]. Oxidation of low density lipoproteins, which are prominent in preeclampsia, is one example of the measurable consequences of increased oxidative stress[46].

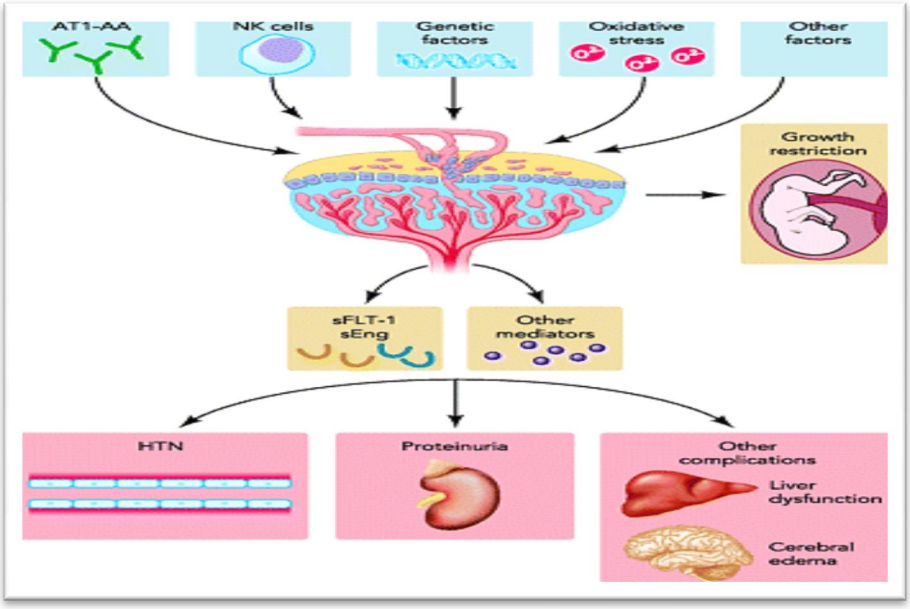
There are several methods of examining lipid peroxidation products in biological samples, but most of them have both low specificity and low sensitivity in terms of measuring changes in free radical status. Among such lipid peroxidation products are the19tiobarbituric acid reactive substances (TBARS), lipid hydroperoxides and the isoprostanes[47-49].There are also end products of oxidative stress, other than lipid peroxidation products, that can be measured as indices of oxidative stress. Direct damage to proteins by peroxidation of the amino acids can also give rise to protein carbonyls,

which may serve as more general biomarkers of oxidative stress [50]. Plasma protein carbonyls have been demonstrated to be elevated in preeclampsia as compared to controls [51].Peroxinitrate, produced by the vasorelaxant nitric oxide (NO) reacting with superoxide anions (produced under conditions of oxidative stress) is also a potential marker of oxidative stress. Peroxinitrite is regarded as a marker of "nitrative stress" which, subsequent to oxidative stress, is seen in the placenta in preeclampsia and diabetes in association with altered placental function [52]. NO is produced by endothelial cells and is also known to react with superoxide anions (produced under conditions of oxidative stress),yielding peroxynitrite that may impair vascular function[53].

ANGIOGENIC FACTORS IN PREECLAMPSIA

Considerable attention has recently been focused on angiogenesis-related factors in the etiology of preeclampsia. It has been postulated that preeclampsia might be a syndrome of angiogenic disorders and that the angiogenic factor like soluble fms-like tyrosine kinase 1(sFlt1) plays a role in the pathophysiology of preeclampsia[25,54]. Growth factors like PlGF (placental growth factor) and VEGF (vascular endothelial growth factor) are necessary for the endothelial function and the angiogenesis and vasculogenesis of early placentation[55].

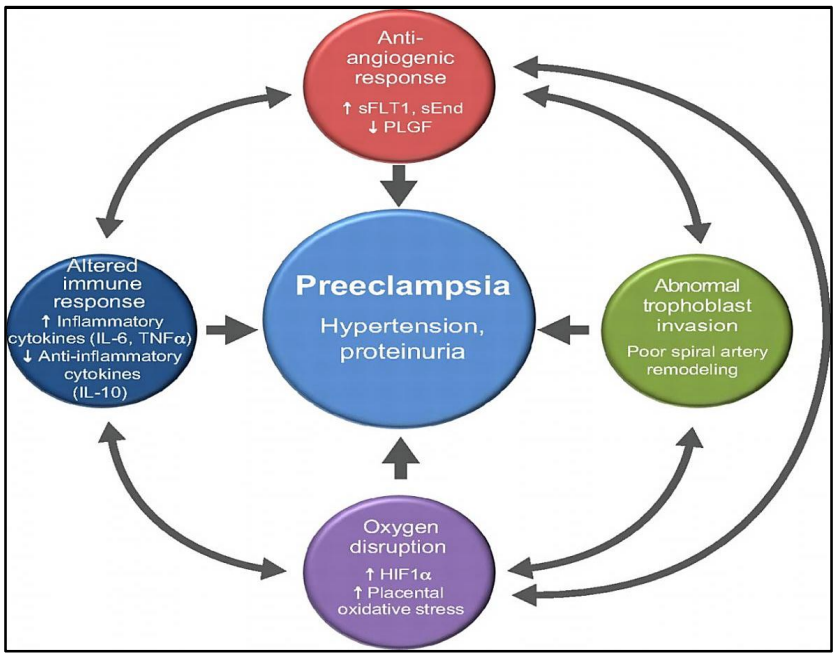
Recently the anti-angiogenic factor sFlt1, which binds soluble VEGF and PlGF and inhibits their effect on the vascular endothelium, was found to be up-regulated in preeclamptic placentas[25]. High serum levels of sFlt have been found in preeclampsia[24,25], and increased sFlt 1concentrations in the serum predict the onset of preeclampsia in the second trimester [23]. A recent Norwegian study also showed that a low rise of maternal PlGF and a high rise of sFlt1 between the first and second trimester are strong predictors of early onset preeclampsia [26].



Summary of the pathogenesis of preeclampsia Immune factors (such as AT1-AA), oxidative stress, NK cell abnormalities, and other factors may cause placental dysfunction, which in turn leads to the release of anti-angiogenic factors (such as sFlt1 and sEng) and other inflammatory mediators to induce hypertension, proteinuria, and other complications of preeclampsia.

Recently, soluble endoglin (sEng) has been found in elevated concentrations in preeclamptic maternal serum[26] sEng and sFlt1 causes endothelial dysfunction in vitro by blocking the angiogenic effects of TGF- and VEGF and can also induce an illness similar to severe preeclampsia in pregnant rats[25,26.]

PATHOGENESIS OF PREECLAMPSIA



Preeclampsia has been rightly termed as the disease of theories and the various pathogenic mechanism involved are as follows

1. Abnormal trophoblastic invasion
2. Inflammation and endotheial cell injury
3. Oxygen disruption
4. Antiangiogenic factors

RISK FACTORS

One out of every three woman who are preeclamptic has a significant risk factor. As per NICE(NATIONAL INSTITUTE OF CLINICAL EXCELLENCE) guidelines, a carefull and detailed history taking should be done at every woman's first antenatal visit in order to ascess her risk level for developing preeclampsia and her subsequent antenatal visits can be planned from the evaluation made.

RISK FACTORS –NICE guidelines

1. Nulliparity
2. History of preeclampsia in a first degree relative like sister/mother
3. Age >40 years
4. Previous obstetric history of preeclampsia
5. BMI(body mass index) >35 in the first visit
6. Preexisting vascular diseases like diabetes and hypertension[56.]
7. Multiple pregnancy

Recently some of these risk factors have been quantified at the booking visit by a systemic review[57 .]

History of preeclampsia in the family signifies the genetic influence of the disease. Preeclampsia is more frequently seen in primigravida patients[58]. The development of preeclampsia in the fetus exposed to the affected paternal antigen suggest a significant immunological influence of the disease .Similarly if this pregnancy is conceived with a partner who had already fathered a preeclamptic pregnancy then the risk of preeclampsia is doubled in this pregnancy[61]. Increased duration of sexual cohabitation and the use of contraceptives of non-barrier methods are known to decrease the risk of preeclampsia[59,60].

If the patients has any associated medical illness involving the CVS or any form of carbohydrate intolerance such as gestational diabetes mellitus or pre-gestational diabetes the risk of preeclampsia is increased in the mother[62,63]. One of the important independent risk factor of preeclampsia is obesity.

Size of the placenta also appears to be an independent risk factor as can be seen in molar pregnancies which are rare causes of early onset preeclampsia. Patients with complicated pregnancies like trisomy chromosomal anomaly in the fetus or the presence of hydrops fetalis also called as mirror syndrome are also known to have an increased risk of preeclampsia. Every one out of five patients who had a previous history

of preeclampsia associated with delivery of the fetus before 37 weeks due to preeclampsia are known to have a recurrence of preeclampsia in the present pregnancy as well.

INVESTIGATIONS

Predictive tests for preeclampsia is divided into biophysical and biochemical test

Biophysical Tests

1. Uterine artery Doppler
2. Blood pressure measurement in early pregnancy
3. Isometric exercise testing
4. Roll over test
5. Angiotensin II sensitivity test

Biochemical and Haematological Test

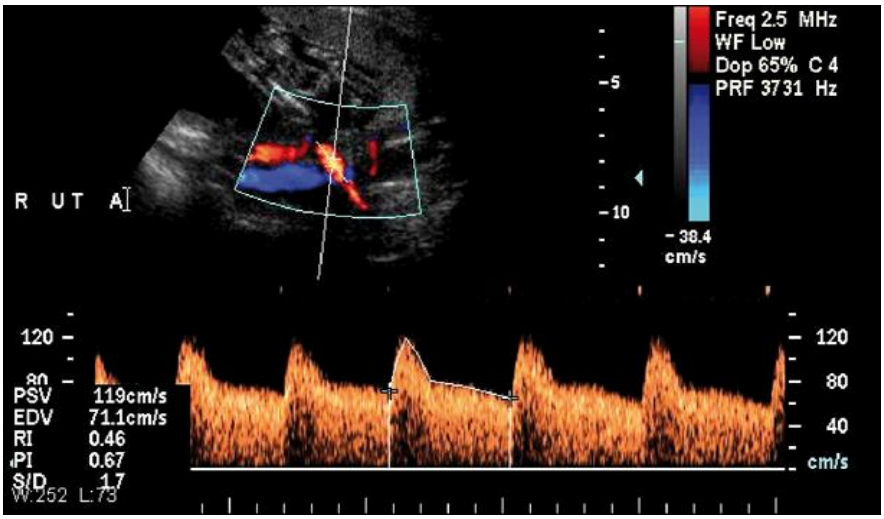
1. Haemoglobin and PCV
2. Serum uric acid
3. Platelet count
4. Beta HCG and alpha fetoprotein
5. sEng(soluble endoglin) and sFlt 1
6. urinary calcium excreation
7. prostacyclin metabolites

Uterine Artery Doppler

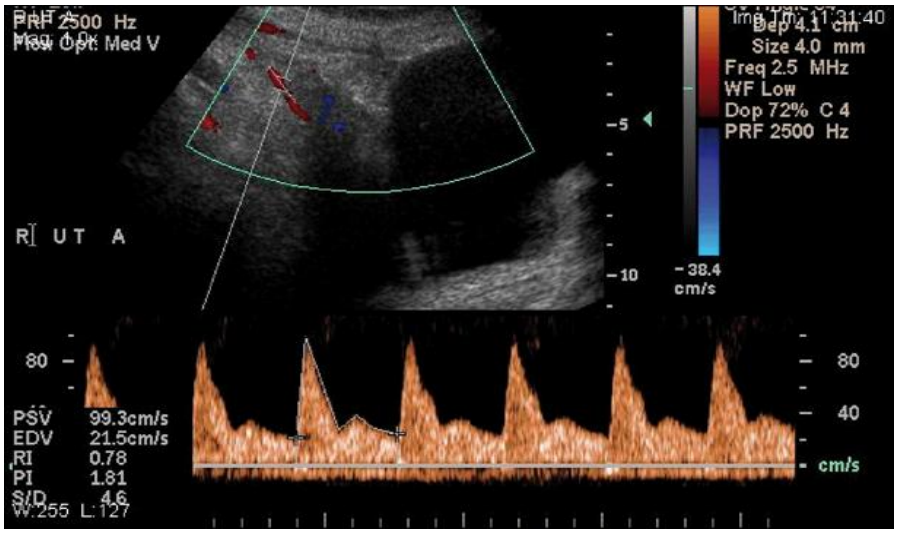
It is a quick and relatively inexpensive test that is usually done around 20 weeks of gestation .ie. around the time anomaly scan is being performed to identify poor placental perfusion ,the main reason for the pathophysiology of the disease process[64].The uterine artery Doppler shows a high resistant circulation with a diastolic notch which is an important predictator of preeclampsia. But due to a delay in the change to a low resistant circulation even in some normal pregnancies the positive predictive value of the test can be improved by performing the test at a later gestational age especially around 24 week which increases the predictive value of the test.

One in every five woman with an abnormal high resistant circulation with a diastolic notch in the uterine artery Doppler performed at around 20 weeks of gestation have an increased risk of developing preeclampsia. Thus Doppler helped to identify high risk woman at an earlier gestational age there by allowing the use of prophylactic therapies to decrease the severity of the disease. However the use of this biophysical test for improving the outcome of the disease process has not yet been established and as clearly stated by NICE who do not recommend the use of Doppler in low risk woman.

Normal “low resistance” uterine artery waveform in the midtrimester.



Abnormal uterine artery vascular resistance, showing increased pulsatility and the presence of an early diastolic notch.



Blood Pressure Measurement In Early Pregnancy

Alteration in the circadian variability are present as early as first trimester in a women at risk of preeclampsia. But due to its subjective and objective variability it is an inaccurate predictor of pre eclampsia[65].

Others like Isometric exercise testing and Roll over test have low predictive value.

Angiotensin Ii Sensitivity Test

A women at risk of developing preeclampsia will respond to less than 8 ng/kg min of angiotensin infusion due to alteration of vessel wall refratoriness. This test is done between 26 – 30 weeks[66].

Biochemical And Hematological Predictors Of Pre Eclampsia

Hemoglobin and hematocrit are poor predictors of preeclampsia as does plasma volume. In chronic hypertension patients serum uric acid and platelets can be measured to identify those patients with a superimposed pre eclampsia but they lack sensitivity and specificity. Beta HCG and alpha fetoprotein measured in the second trimester increases the risk of preeclampsia two fold. This is due to the pathological process taking

place at the utero- placental interface. But the positive predictive value for beta HCG and alpha fetoprotein is very low and hence not clinically practicable.

Endothelial markers such as soluble fms like tyrosine kinase 1, placental growth factor, vascular endothelial growth factor and soluble endolglin are also predictors of preeclampsia. These act by inhibiting the nitric oxide action of vasodilatation. However these are also seen in normal women thereby limiting the clinical usefulness of these markers.

Urinary Kallikrein / creatinine ratio of < 170 between 16 and 20 weeks having sensitivity of 70 % and specificity of 86 % predicts the future development of preeclampsia.

Urinary calcium excretion is reduced in preeclampsia due to increased proximal and distal tubular calcium re-absorption. A urinary calcium level of<12mg/dl has sensitivity of 91 %.Antithrombin III is reduced in preeclampsia which correlates with maternal and perinatal outcome.

Serum fibronectin levels are elevated in preeclampsia. In preeclampsia Pregnancy associated plasma protein A(PAPP-A) is

reduced in first trimester. Both these markers are associated with two fold risk of developing preeclampsia later in pregnancy.

PREVENTION OF PREECLAMPSIA

Numerous interventions have been tried to reduce the severity of preeclampsia.

Diet and exercise

Various studies investigating the role of protein restriction or supplementation, aerobic exercises, magnesium and zinc supplementation, and decreased salt intake have given conflicting results. But randomized controlled studies have reported minimal or no benefit[67].

Calcium supplementation

A systematic review has found 30 % reduction in the risk of preeclampsia with an intake of 1 gram calcium per day[68]. This effect is greatest for women at high risk of developing preeclampsia and for those with previous low calcium intake. But this reduction did not have an effect on still birth and neo natal mortality rate.

Aspirin and ant-platelet agents

CLASP (Collobarative low dose aspirin study), a large randominised control study reported a reduction of preeclampsia in 12% but this was insignificant. However some benefit was seen in a small subset of women who were at high risk of developing preeclampsia when aspirin was started early in the pregnancy. Also it showed that low dose aspirin was safe for the fetus. By comparing the results of 43 trials antiplatelet agents and low dose aspirin were found to decrease the risk of preeclampsia by 19%. Similarly an 8% reduction in preterm births and a 14% reduction in perinatal mortality were seen in patients on a prophylactic dose of aspirin[69,70].

Use of fish oils with N3 fatty acid which inhibit platelet thromboxaneA2 did not show a significant reduction in preeclampsia. Since oxidative stress is an important factor to the development of the disease process vitamin C and E supplementation in the dosage of 1000mg and vitamin D 400 IU, in the second half of pregnancy demonstrates more than a 50% reduction in the risk of preeclampsia[71]

CLINICAL FEATURES

The classic triad of preeclampsia are

- Hypertension, defined by Systolic pressure > 140mm of Hg and Diastolic > 90mm of Hg.
- Proteinuria - 0.3 g in a 24-h urine specimen or protein to creatinine ratio of >0.30.
- Edema, though non-specific, sudden onset of gross edema of the hands and face, is a definitive feature in the diagnosis of preeclampsia.

The clinical presentation of preeclampsia varies widely,even without the presence of all three classical symptoms. It has been reported that 10% of cases of gestational hypertension without significant proteinuria have progressed to severe preeclampsia[72]. Hence a strict follow-up of patients with isolated hypertension is necessary and lifesaving in the antenatal period[73,74].

Another study by Lafayette et al ., has showed significant reduction in the GFR in preeclampsia patients while the renal plasma flow and oncotic pressure remains normal[75].

Some of the serious complications include pulmonary edema, seizures, acute kidney injury, abruption placentae and in extreme cases, HELLP syndrome has been reported. The triad - Hemolysis, Elevated Liver enzymes[76] and Low Platelets is associated with serious maternal and neonatal complications.

The pathogenesis of preeclampsia- subtle vascular damage and persistent endothelial dysfunction is the reason for a variety of related complications. In the long term about 20% of preeclampsia patients develop either hypertension or microalbuminuria within 7 years of the last pregnancy. Other serious long term complications include Renal - an increased risk of End Stage Renal Disease (ESRD), Cardiovascular – increased TGL, LDL, Sr.Cholesterol and BMI values and cerebrovascular diseases.

PREECLAMPSIA AND FETAL ASPECTS

The fetal well-being and development in utero during pregnancy is dependent on both the fetal genes and the intrauterine environment. The intrauterine environment is mainly mediated through the placenta, which serves all the needs of the infant, including nutritional and oxygen supply, disposing of waste products as well as serving hormonal support for fetal

growth and development. In pregnancies complicated with preeclampsia, the consequences for the fetus may be due to both prematurity, like lack of lung maturation and growth restriction, as well as the long term consequences for the infant, like increased risk of developing cardiovascular disease, dying from stroke and developing hypertension in adulthood[77].

In preeclampsia the final common pathway is believed to be due to maternal endothelial dysfunction[10,11.] This endothelial dysfunction is believed to be due to the release of certain agents into the maternal circulation by the hypoxic placenta.[78-81]. Prematurity has been associated with increased oxidative stress[82-84], and oxidative stress has also been implicated in several of the diseases of prematurity such as bronchopulmonary dysplasia, intraventricular hemorrhage and necrotizing enterocolitis[84-86]. 8-isoprostane, an acknowledged marker of oxidative stress, has been demonstrated to be elevated in plasma of preterm infants compared to term infants in only one study [87], and also low concentrations of antioxidants in the fetal circulation have been demonstrated, possibly representing infants more vulnerable to oxidative injury[88-90]. Since preeclampsia is associated with increased oxidative stress both in the maternal circulation and in the placenta, it is of interest

to explore whether this imbalance between pro-and antioxidants also is present in the circulation of an infant born to a preeclamptic mother.

MANAGEMENT OF PREECLAMPSIA

It is concerned with the maximal prolongation of the duration of pregnancy without affecting the maternal or fetal well being.

- Admission and rest

  One study compared the effect of bed rest with that of liberal ambulation on the physiological changes of preeclampsia. Renal function had no improvement but the placental function had some signs of improvement.

- BP monitoring[91,92]

- Urine albumin and urine output monitoring daily

- Fetal monitoring

- Watch for signs of imminent eclampsia

- Antihypertensive therapy

Oral labetalol is a beta blocker and is the drug of choice in preeclampsia[93].It is started when the BP persistently stays above

150/100mm/Hg.It is started in dosage of 200mg BD upto a maximum dose of 2.4 g. However it can reduce the cardiac output to the uterus leading to IUGR. Other drugs such as Nifedipine ,a calcium channel blocker can be given in dosage of 5-10mg BD. It acts by blocking the calcium influx into the smooth muscle cells, interfering with the excitation – contraction coupling of the muscle. Side effects include headache, facial flushing and warm , sweaty extremities.

- Timing of delivery

About 50% of patients with mild preeclampsia will develop severe preeclampsia remote from term. They are even at a risk of developing eclampsia suddenly without warning and with minimal BP elevations. Other such serious complication is abruption placenta. But both these risks constitute <1%. In mild preeclampsia the use of antihypertensive agents helps to reduce the progression to severe preeclampsia but offers no benefit to the fetus.

Pregnancy is terminated at 38 weeks by inducing labour or by an elective cesarean section if cephalopelvic disproportion is seen or much earlier or if it progresses to severe preeclampsia.

If the disease progresses from mild to severe or if there is an indication of fetal compromise like oligohydramnious, non-reassuring fetal heart rate pattern or intrauterine growth restriction the patient must be planned for delivery.

Serial laboratory evaluation of platelet count, haemoglobin ,haematocrit, serum uric acid ,blood urea & creatinine , liver enzymes like SGOT, SGPT, SAP, urine for albumin must be done weekly to monitor for worsening of the disease. Also patients are instructed to monitor daily fetal kick count and undergo twice weekly non stress test and amniotic fluid index, weekly biophysical profile . Doppler and ultrasound for fetal growth should be done once in every 3-4 weeks . Once the diagnosis is established the only cure is termination of the pregnancy. The indicators for termination of pregnancy are as follows

- Persistently elevated BP of 160/110 mm/Hg
- Proteinuria >5 grams in 24 hour
- Headache &blurring of vision
- Epigastric pain
- HELLP syndrome
- Compromised renal function test

- Oliguria <400 ml in 24 hours
- Pulmonary edema
- Abruption placenta
- Eclampsia
- IUGR with abnormal Doppler
- Evidence of fetal distress like non reactive CTG, biophysical profile of <4 on two occasions ,four hours apart, severe IUGR <5 th percentile for gestational age and severe oligohydramnious.
- Fetal death ,rupture of membrane, spontaneous labour
- Gestational age >34 weeks in cases of severe preeclampsia

MANAGEMENT OF SEVERE PREECLAMPSIA

Before 24 weeks of gestation termination of pregnancy is recommended. The preferred mode is induction of labour if the BP levels are well controlled without any toxic clinical appearance.

The management of severe preeclampsia in mid trimester (25 – 32 weeks)- Steroids were given to hasten lung maturity and delivery can be delayed after 24 hours if possible. During this period extensive maternal

and fetal surveillance should be done. While conservative management may be associated with an increase in maternal complications, aggressive management in delivery may result in neonatal mortality.

Expectant management in severe preeclampsia before 32 – 34 weeks in patients having only increased proteinuria > 5 gram in 24 hours is justified to prolong the gestation. Patients > 34 weeks should be delivered. In these patients diastolic BP should be maintained between 95- 105 mm hg. It is always better to induce the labour provided there are no contraindications such as severe IUGR or contracted pelvis. However the threshold for cesarean section should be kept low. Large amount of peritoneal fluid is found during cesarean.

Objectives in the management :

- To prevent complications such as pulmonary edema,renal failure, abruption and cardiovascular complications .
- To prevent convulsions as it is associated with 10 times increased risk of maternal and perinatal mortality.
- To deliver a healthy baby with least maternal morbidity.

As volume contraction is poorly tolerated in these patients , it is prudent to take necessary steps to minimize blood loss. Cross matched blood and blood products should be kept ready. She should be assisted in second stage of labour inorder to prevent the rise in BP during each uterine contraction. Methyergometrine is contraindicated .

Appropriate steps should be taken to minimize postpartum hemorrhage particularly in patients receiving MgSO4.

Antihypertensives in severe pre eclampsia

Intravenous hydralazine , intravenous labetalol and oral nifidepine are the commonly used agents to control acute ,very high BP. Hydralazine is given at a dosage of 5 – 10 mg intravenous every 20 minutes upto a maximum dosage of 30 mg. It may cause headache, nausea , vomiting and maternal hypotension.

Labetalol is given at 10 – 20 mg iv , then 40 – 80 mg every 10 minutes upto a maximum dosage of 300 mg , a continuous infusion of 1-2 mg can also be used. It has a favourable side effect profile except for the fetal bradycardia it causes

Sublingual nifedipine is not used due to fear of severe hypotension. Oral nifedipine is given at a dosage of 10 mg , repeated every 30 minutes, followed by 10 – 20 mg every 4 hours, upto a maximum of 240mg. In conjunction with MgSO4 it may cause profound hypotension and hence it should be carefully used in coronary heart disease patients.

Intravenous sodium nitroprusside can be used in refractory hypertension with fetal cyanide poisoning being the risk on prolonged treatment. Usually hypertension resolves 24 hours after delivery. If it persists antihypertensives are continued. A diagnosis of chronic hypertension should be considered if hypertension persists for more than 6 weeks postpartum.

The HELLP Syndrome

Weinstein coined the acronym HELLP in 1982 to aid clinicians in recognizing this group of patients with remarkable hepatic involvement by severe preeclampsia (Weinstein 1982). Indeed the features of the HELLP syndrome are very similar and sometimes difficult to distinguish from those of other microangiopathic syndromes that may develop in late pregnancy such as hemolytic uremic syndrome and thrombotic

thrombocytopenic purpura. The latter disorders are characterized by vascular endothelial cell damage, elicited by unknown antigens or toxic substances. Differentiation of these disorders from the HELLP syndrome is important because the latter is treated by urgent delivery, whereas hemolytic uremic syndrome or thrombotic thrombocytopenic purpura may require plasma infusion or plasmapheresis.

Sibai has noted that most patients with the HELLP syndrome present preterm with hypertension and proteinuria and report epigastric and upper-right quadrant pain (1990). Around 30%of HELLP cases develop postpartum. The following criteria are used to establish the diagnosis of the HELLP syndrome(Sibai 2002):

- Hemolysis
- Abnormal peripheral blood smear
- Increased bilirubin ≥1.2mg/dl
- Increased lactate dehydrogenase (LDH) >600 IU/I
- Elevated liver enzymes
- Increased SGOT ≥ 72 IU/I
- Increased LDH>600 IU/I
- Thrombocytopenias
- Platelet count < 1 00,000 cumm

Most obstetricians consider the development of HELLP syndrome as an indication for urgent delivery, because the laboratory values can reach life threatening levels with severe thrombocytopenia and elevated liver enzymes. Liver rupture is rare, with a high mortality rate requiring large amounts blood and blood products transfusions and laparotomy, drainage and packing.

The maternal condition is first assessed and stabilized. Blood investigations are taken to assess the biochemical severity. Antihypertensives are started. MgSO4 started as seizure prophylaxis[94]. The fetal condition is also assessed and the decision to deliver immediately is decided upon.

High dose corticosteroids (10 mg iv dexamethasone every 6 hours for two doses, followed by 6 mg every 6 hours two doses) are given to improve maternal condition as steroids appear to alter the final steps of endothelial cell disruption.

The HELLP syndrome is classified based on platelet count as Class 1 –(< 50,000), Class 2 – (50,000 – 1 lakh) and Class 3 – (1 lakh-1,50,000). They help to predict the rapidity of postpartum delivery. Platelet transfusions are indicated, either before or after delivery, in the

presence of significant bleeding from puncture sites and so on. It is necessary to transfuse when platelet count is less than 20,000.

Thrombophilia and Preeclampsia

A combination of aspirin and heparin or low molecular weight heparin is effective in recurrent fetal loss in the APLA syndrome and the other inherited thrombophilias.

Seizure Prophylaxis

MgSO4 can be given in severe preeclamptic patients to prevent convulsions[95].

Anesthesia and analgesia

Continuous epidural analgesia is very useful for vaginal and operative delivery[96]. It offers dual benefits of analgesia and stabilization of BP. It prevents the release of catecholamines during uterine contractions. It abolishes excessive bearing down during the second stage labour. Patient should be adequately preloaded , fully conscious and coagulopathy must be excluded before inserting the epidural catheter.

If general anesthesia is indicated , the following risk should be kept in mind.

1) Laryngeal edema which makes intubation difficult
2) The pressor response to intubation precipitating pulmonary edema or arrhythmia.
3) Risk of aspiration
4) Decrease in uteroplacental flow which may hazardous to severe IUGR fetus
5) Neuromuscular blockade

Postnatal assessment

Persistence of hypertension or proteinuria after 6 weeks necessitates futher work up[97]. Pregnancy should be advised only if BP and renal function tests return to normal. It is necessary to councel the patients for early booking of future pregnancies. The advantageinclude early administration of aspirin and detection of preeclampsia at the earliest.

RECURRENCE

The risk of recurrent preeclampsia was 3.4 %. The risk of recurrent hypertension without proteinuria was even more higher about 25%. Pre eclampsia developing in second trimester during first pregnancy proposes 60 % risk of recurrence in second pregnancy. Change in paternity also imparts increased risk of preeclampsia in multipara. Patients with preeclampsia are more prone to vascular disease and chronic hypertension later in life.

AIM OF THE STUDY

AIM OF THE STUDY

- To study the ability of immediate postpartum curettage on accelerated recovery from severe preeclampsia

- Effect on resolution of laboratory indices

MATERIALS AND METHODS

MATERIALS AND METHODS

SETTING	RSRM Lying in hospital, Stanley, Chennai
DESIGN OF STUDY	Randomnised Prospective control study
PERIOD OF STUDY	1 year
STUDY POPULATION	200 pregnant patients >28 weeks with severe preeclampsia admitted in RSRM Lying in hospital

INCLUSION CRITERIA

28 Weeks and above gestational age with either of the three features of Severe PIH like:

1. BP of 160/110 and above at time of admisssion.
2. 2+ proteinuria in semi quantitative analysis.
3. less than 400 ml of urine output over 24 hours.

As these features usually don't exist consistently and concurrently in all patients these additional criteria are taken such as altered consciousness, headache, blurred vision, epigastric or right upper quadrant pain, impaired liver function of unclear etiology, thrombocytopenia.

EXCLUSION CRITERIA

1. < 28 Weeks of Gestation
2. Mild PIH
3. Cardiovascular and Renal Disorder
4. Hypertension before Pregnancy
5. Convulsion Disorder and Liver Disorders
6. Eclampsia.

METHODOLOGY

Sample size:

200 pregnant women fulfilling the inclusion criteria of which100 undergoing Curettage and 100 not undergoing curettage. As per the hospital records during, the last 5 years there were around two hundred

cases of severe preeclampsia every year in RSRM LYING IN HOSPITAL. With reference to that, two hundred cases were selected for the present study as a convenience sampling. No formula was used for sample size

Sampling Technique: Randomization

Pregnant women fulfilling the criteria will be selected by randomization such as those with even numbers in intervention group and those with odd numbers in the non intervention group. Thus 200 respondants will be selected for study purpose. The method of randomisation is computer based random numbers.

METHOD OF COLLECTION OF DATA

The admission tests involve BP, proteinuria, platelet count, LFT, RFT measurement of urine output. As a routine, the placental location is noted as per USG to note the site for curettage. Data will be collected on patients status regarding

- Cerebral and visual disturbances, headache and blurring of vision
- Epigastric pain.
- Impaired liver function test of unclear etiology
- Thrombocytopenia.

The study group will undergo curettage at vaginal or caesarean delivery. At caesarean the presumed decidua basalis will be curetted with large blunt curette. Patients who delivered vaginally will be curetted under I.V sedation using large blunt curette . This study is done on postpartum patients.

Post-partum surveillance will be done- for 48 hours. With 4 th hourly BP recording, 4 th hourly urine output, platelet count, LFT and RFT 24 hourly

All medications given are noted. Patients stay in CCU/Labour ward is noted.

Patients are shifted to postoperative ward from CCU only if the following criteria is fulfilled.

CRITERIA-

a) adequate diuresis(>100 ml/hr for atleast two consecutive hours)
b) BP <150/100mm/Hg
c) Rising platelet count of >50,000/ml

OBSERVATIONAL

ANALYSIS

OBSERVATIONAND ANALYSIS

TABULAR COLUMN 1 - AGE AND GESTAIONAL AGE

	Group	N	Mean	Std. Deviation	P value
Age in years	Control	100	24.18	3.702	0.581
	Cases	100	23.89	3.712	
Gestational age(wks)	Control	100	36.67	1.843	0.280
	Cases	100	36.37	2.068	

* The mean age and mean gestational age on admission was 24 years and 36 -37weeksfor both the control and study group. They are statistically insignificant (NS) and hence both the control and study group are comparable.

TABULAR COLUMN 2- PARITY

			Group		Total	
			Control	**Cases**		
Parity	Primi	Count	58	57	115	0.886
		% within Parity	50.4%	49.6%	100.0%	
		% within Group	58.0%	57.0%	57.5%	
	Multi	Count	42	43	85	
		% within Parity	49.4%	50.6%	100.0%	
		% within Group	42.0%	43.0%	42.5%	
Total		Count	100	100	200	
		% within Parity	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

***** Severe preeclampsia most commonly occurred in primigravida constituting about 58% in control group and 57% in the study group with a P value of .886 (NS).

TABULAR COLUMN 3-MODE OF TERMINATION

			Group		Total	P
			Control	**Cases**		
Mode of termination	LSCS	Count	60	57	117	.667
		% within Mode of termination	51.3%	48.7%	100.0%	
		% within Group	60.0%	57.0%	58.5%	
	Normal	Count	40	43	83	
		% within Mode of termination	48.2%	51.8%	100.0%	
		% within Group	40.0%	43.0%	41.5%	
Total		Count	100	100	200	
		% within Mode of termination	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

* In this study about 57% in the study group and 60% in the control group underwent LSCS while the remaining had a normal vaginal delivery with a P value of .667 (NS).

TABULAR COLUMN 4-URINE ALBUMIN ON ADMISSION

			Group		Total	P
			Control	74**Cases**		
Urine albumin – Pre	1+	Count	42	32	74	
		% within Urine albumin – Pre	56.8%	43.2%	100.0%	
		% within Group	42.0%	32.0%	37.0%	
	2+	Count	56	55	111	
		% within Urine albumin – Pre	50.5%	49.5%	100.0%	
		% within Group	56.0%	55.0%	55.5%	
	3+	Count	12	13	25	.120
		% within Urine albumin – Pre	83.3%	86.7%	100.0%	
		% within Group	12.0%	13.0%	12.5%	
Total		Count	100	100	200	
		% within Urine albumin – Pre	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

* On admission urine albumin was comparable in both the study and control group with a P value of .120(NS)

TABULAR COLUMN 5-Tablet LABETALOL ON ADMISSION

			Group		Total	P
			Control	**Cases**		
Labetalol - Pre	1	Count	19	13	32	.121
		% within Labetalol – Pre	68.1%	71.9%	100.0%	
		% within Group	19.0%	13.0%	32.0%	
	2	Count	81	87	168	
		% within Labetalol – Pre	49.2%	45.8%	100.0%	
		% within Group	81.0%	87.0%	169.0%	
Total		Count	100	100	200	
		% within Labetalol – Pre	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

TABULAR COLUMN 6-Tablet NIFEDIPINE ON ADMISSION

			Group		Total	P
			Control	Cases		
Nifidepine – Pre	1	Count	59	53	112	.232
		% within Nifidepine – Pre	44.5%	55.5%	100.0%	
		% within Group	59.0%	53.0%	112.0%	
	2	Count	41	47	88	
		% within Nifidepine – Pre	45.9%	54.1%	100.0%	
		% within Group	41.0%	47.0%	88.0%	
Total		Count	100	100	200	
		% within Ni fidepine – Pre	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

* On admission antihypertensives like Tab Labetalol and Tab Nifedipine was given equally to both study and control group with a P value of .121 and <.232(NS)

TABULAR COLUMN 7-LABORATORY INVESTATIGATIONS ON ADMISSION

	Group	N	Mean	Std. Deviation	Std. Error Mean	p Va;ue
SBP – Pre	Control	100	158.50	6.416	.642	.204
	Cases	100	156.80	11.710	1.171	
DBP – Pre	Control	100	106.60	7.278	.728	.605
	Cases	100	106.10	6.340	.634	
Urine output - Pre	Control	100	85.00	34.304	3.430	.687
	Cases	100	83.10	32.183	3.218	
Platelet count (lakhs/litre) – Pre	Control	100	1.435	.2463	.0246	.310
	Cases	100	1.398	.2674	.0267	
Bl.Urea (mg/dl) - Pre	Control	100	23.90	6.209	.621	.833
	Cases	100	23.73	5.120	.512	
S.Creatinine (mg/dl) – Pre	Control	100	.603	.1566	.0157	.894
	Cases	100	.600	.1627	.0163	
Uric acid (mg/dl) - Pre	Control	100	6.832	.9125	.0913	.410
	Cases	100	6.925	.9889	.0989	
SGOT(mEq/L) - Pre	Control	100	42.34	15.574	1.557	.964
	Cases	100	42.22	21.294	2.129	
SGPT(mEq/L) - Pre	Control	100	42.26	16.673	1.667	.774
	Cases	100	43.12	24.814	2.481	
SAP(mEq/L) - Pre	Control	100	97.52	25.924	2.592	.317
	Cases	100	102.23	39.101	3.910	

On admission the laboratory values of platelet count, urine output, blood urea, serum creatine, SGOT, SGOT and SAP were found to be within normal limits except for a very few patients with elevated liver enzymes and moderately reduced urine output in both the control group and study group. The mean of each value in both the study and control group are as follows:

SBP – Pre	Control	158.50
	Cases	156.80
DBP - Pre	Control	106.60
	Cases	106.10
Urine output – Pre	Control	85.00
	Case	83.10
Platelet count (lakhs/litre) – Pre	Control	1.435
	Cases	1.398
Bl.Urea (mg/dl) - Pre	Control	23.90
	Cases	23.73
S.Creatinine (mg/dl) – Pre	Control	.603
	Cases	.600

Uric acid (mg/dl) - Pre	Control	6.832
	Cases	6.925
SGOT(mEq/L) - Pre	Control	42.34
	Cases	42.22
SGPT(mEq/L) - Pre	Control	42.26
	Cases	43.12
SAP(mEq/L) – Pre	Control	97.52
	Cases	102.23

Hence all these values have a P value of >.05 (NS) making them comparable with each other.

TABULAR COLUMN 8-INVESTIGATION REPORTS ON DAY 1

	Group	N	Mean	Std. Deviation	Std. Error Mean	P value
SBP - Day 1	Control	100	152.70	6.795	.679	.000
	Cases	100	147.40	6.609	.661	
DBP - Day 1	Control	100	99.90	5.773	.577	.000
	Cases	100	96.00	4.924	.492	
Urine output - Day 1	Control	100	91.00	31.286	3.129	.000
	Cases	100	112.50	28.758	2.876	
Platelet count (lakhs/litre) - Day 1	Control	100	1.448	.2359	.0236	.138
	Cases	100	1.495	.2100	.0210	
Bl.Urea (mg/dl) - Day 1	Control	100	23.89	5.896	.590	.060
	Cases	100	22.57	3.740	.374	
S.Creatinine (mg/dl) - Day 1	Control	100	.586	.1429	.0143	.103
	Cases	100	.554	.1337	.0134	
Uric acid (mg/dl) - Day 1	Control	100	6.657	.8059	.0806	.000
	Cases	100	6.091	.8545	.0854	
SGOT(mEq/L) - Day 1	Control	100	41.64	14.761	1.476	.087
	Cases	100	38.03	14.906	1.491	
SGPT(mEq/L) - Day 1	Control	100	41.07	14.869	1.487	.205
	Cases	100	38.25	16.473	1.647	
SAP(mEq/L) - Day 1	Control	100	93.53	17.400	1.740	.433
	Cases	100	91.33	21.932	2.193	

* On the first postnatal /postoperative day there is a significant fall of BP and Serum Uric Acid and a moderate increase in urine output in the study group who underwent curettage than those patients in the control group

TABULAR COLUMN 9-INVESTIGATION REPORTS ON DAY2

	Group	N	Mean	Std. Deviation	Std. Error Mean	P
SBP - Day 2	Control	100	148.10	5.979	.598	<.001
	Cases	100	137.20	5.333	.533	
DBP - Day 2	Control	100	95.60	5.379	.538	<.001
	Cases	100	89.10	2.876	.288	
Urine output - Day 2	Control	100	96.50	29.521	2.952	<.001
	Cases	100	130.50	28.333	2.833	
Platelet count (lakhs/lt) - Day 2	Control	100	1.495	.2508	.0251	.021
	Cases	100	1.570	.2042	.0204	
Bl.Urea (mg/dl) - Day 2	Control	100	23.38	5.763	.576	.002
	Cases	100	21.32	3.390	.339	
S.Creatinine (mg/dl) - Day 2	Control	100	.572	.1450	.0145	.010
	Cases	100	.521	.1328	.0133	
Uric acid (mg/dl) - Day 2	Control	100	6.536	.7713	.0771	<.001
	Cases	100	5.243	.6663	.0666	
SGOT(mEq/L) - Day 2	Control	100	41.30	14.487	1.449	<.001
	Cases	100	34.00	6.566	.657	
SGPT(mEq/L) - Day 2	Control	100	40.56	14.445	1.444	<.001
	Cases	100	33.63	6.851	.685	
SAP(mEq/L) - Day 2	Control	100	90.93	12.608	1.261	<.001
	Cases	100	84.96	9.997	1.000	

Mean and P Value of Each Investigations on Day 2

SBP (mm/Hg)		P value
Control	148.10	<.001
Cases	137.20	

DBP (mm/Hg)		
Control	95.60	<.001
Cases	89.10	
Urine output (ml/Hr)		
Control	96.5	<.001
Cases	130.50	
Platelet count(lakhs/cumm)		
Control	1.495	.021
Cases	1.570	
Bl.Urea (mg/dl)		
Control	23.38	.002
Cases	21.32	
S.Creatinine (mg/dl)		
Control	.572	.010
Cases	.521	
Uric acid (mg/dl)		
Control	6.536	<.001
Cases	5.243	

SGOT(mEq/L)		
Control	41.30	<.001
Cases	34.00	
SGPT(mEq/L)		
Control	40.56	<.001
Cases	33.63	
SAP(mEq/L)		
Control	90.93	<.001
Cases	84.96	

There is a significant decrease in BP, blood urea , serum creatinine, uric acid and liver enzymes with subsequent increase in urine output in patients undergoing postpartum curettage ,except for platelet count which was statistically insignificant.

TABULAR COLUMN 10-Tablet LABETALOL DAY 2

			Group		Total	P
			Control	**Cases**		
Labetalol - Day 2	1	Count	0	2	2	
		% within Labetalol - Day 2	.0%	100.0%	100.0%	
		% within Group	.0%	2.0%	1.0%	
	2	Count	100	98	198	
		% within Labetalol - Day 2	50.5%	49.5%	100.0%	.155
		% within Group	100.0%	98.0%	99.0%	
Total		Count	100	100	200	
		% within Labetalol - Day 2	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

TABULAR COLUMN 11-Tablet NIFEDIPINE DAY 2

			Group		Total	P
			Control	Cases		
Nifidepine - Day 2	0	Count	16	88	104	
		% within Nifidepine - Day 2	15.4%	84.6%	100.0%	<.001
		% within Group	16.0%	88.0%	52.0%	
	1	Count	30	12	42	
		% within Nifidepine - Day 2	71.4%	28.6%	100.0%	
		% within Group	30.0%	12.0%	21.0%	
	2	Count	54	0	54	
		% within Nifidepine - Day 2	100.0%	.0%	100.0%	
		% within Group	54.0%	.0%	27.0%	
Total		Count	100	100	200	
		% within Nifidepine - Day 2	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

* There is no significant decrease in the dosage of tablet labetalol while there is a significant decrease in the dosage of tablet nifedipine (<.001) With 88% of patients in study group having stopped tablet nifedipine

TABULAR COLUMN 12- COMPARISON OF LAB VALUES ON ADMISSION AND ON DAY2 IN CONTROL GROUP

		Mean	N	Std. Deviation	Std. Error Mean	P
Pair 1	SBP – Pre	158.50	100	6.416	.642	<.001
	SBP - Day 2	148.10	100	5.979	.598	
Pair 2	DBP – Pre	106.60	100	7.278	.728	<.001
	DBP - Day 2	95.60	100	5.379	.538	
Pair 3	Urine output – Pre	85.00	100	34.304	3.430	.004
	Urine output - Day 2	96.50	100	29.521	2.952	
Pair 4	Platelet count (lakhs/litre) – Pre	1.435	100	.2463	.0246	<.001
	Platelet count (lakhs/litre) - Day 2	1.495	100	.2508	.0251	
Pair 5	Bl.Urea (mg/dl) – Pre	23.90	100	6.209	.621	.001
	Bl.Urea (mg/dl) - Day 2	23.38	100	5.763	.576	
Pair 6	S.Creatinine (mg/dl) –Pre	.603	100	.1566	.0157	<.001
	S.Creatinine (mg/dl) - Day 2	.572	100	.1450	.0145	
Pair 7	Uric acid (mg/dl) – Pre	6.832	100	.9125	.0913	<.001
	Uric acid (mg/dl) - Day 2	6.536	100	.7713	.0771	
Pair 8	SGOT(mEq/L) – Pre	42.34	100	15.574	1.557	<.001
	SGOT(mEq/L) - Day 2	41.30	100	14.487	1.449	
Pair 9	SGPT(mEq/L) – Pre	42.26	100	16.673	1.667	<.001
	SGPT(mEq/L) - Day 2	40.56	100	14.445	1.444	
Pair 10	SAP(mEq/L) – Pre	97.52	100	25.924	2.592	<.001
	SAP(mEq/L) - Day 2	90.93	100	12.608	1.261	

* There is a significant decrease in BP, urea ,creatinine, uric acid and liver enzymes with increase in urine output and platelet count in the control group.

TABULAR COLUMN 13-COMPARISON OF LAB VALUES IN STUDY GROUP ON ADMISSION AND DAY2

		Mean	N	Std. Deviation	Std. Error Mean	P
Pair 1	SBP – Pre	158.50	100	6.416	.642	<.001
	SBP - Day 2	148.10	100	5.979	.598	
Pair 2	DBP – Pre	106.60	100	7.278	.728	<.001
	DBP - Day 2	95.60	100	5.379	.538	
Pair 3	Urine output – Pre	85.00	100	34.304	3.430	.004
	Urine output - Day 2	96.50	100	29.521	2.952	
Pair 4	Platelet count (lakhs/litre) – Pre	1.435	100	.2463	.0246	<.001
	Platelet count (lakhs/litre) - Day 2	1.495	100	.2508	.0251	
Pair 5	Bl.Urea (mg/dl) – Pre	23.90	100	6.209	.621	.001
	Bl.Urea (mg/dl) - Day 2	23.38	100	5.763	.576	
Pair 6	S.Creatinine (mg/dl) – Pre	.603	100	.1566	.0157	<.001
	S.Creatinine (mg/dl) - Day 2	.572	100	.1450	.0145	
Pair 7	Uric acid (mg/dl) – Pre	6.832	100	.9125	.0913	<.001
	Uric acid (mg/dl) - Day 2	6.536	100	.7713	.0771	
Pair 8	SGOT(mEq/L) – Pre	42.34	100	15.574	1.557	<.001
	SGOT(mEq/L) - Day 2	41.30	100	14.487	1.449	
Pair 9	SGPT(mEq/L) – Pre	42.26	100	16.673	1.667	<.001
	SGPT(mEq/L) - Day 2	40.56	100	14.445	1.444	
Pair 10	SAP(mEq/L) – Pre	97.52	100	25.924	2.592	<.001
	SAP(mEq/L) - Day 2	90.93	100	12.608	1.261	

* Similar to control group the study group also shows a significant decrease in BP, urea, creatinine, uric acid and liver enzymes with significant increase in urine output and platelet count.

TABULAR COLUMN 14- URINE ALBUMIN FOR CONTROL AND STUDY GROUP ON ADMISSION AND ON DAY 2

Group				Urine albumin - Day 2			Total	P
				Trace	Mild	Moderate		
Control	Urine albumin – Pre	Mild	Count	19	23	0	42	<.001
			% within Urine albumin - Pre	45.2%	54.8%	.0%	100.0%	
			% within Urine albumin - Day 2	76.0%	37.7%	.0%	42.0%	
		Moderate	Count	6	38	12	56	<.001
			% within Urine albumin - Pre	10.7%	67.9%	21.4%	100.0%	
			% within Urine albumin - Day 2	24.0%	62.3%	85.7%	56.0%	
		Severe	Count	0	0	2	2	<.001
			% within Urine albumin – Pre	.0%	.0%	100.0%	100.0%	
			% within Urine albumin - Day 2	.0%	.0%	14.3%	2.0%	
	Total		Count	25	61	14	100	
			% within Urine albumin - Pre	25.0%	61.0%	14.0%	100.0%	
			% within Urine albumin - Day 2	100.0%	100.0%	100.0%	100.0%	
Cases	Urine albumin – Pre	Mild	Count	28	4	0	32	<.001
			% within Urine albumin – Pre	87.5%	12.5%	.0%	100.0%	
			% within Urine albumin - Day 2	48.3%	10.0%	.0%	32.0%	
		Moderate	Count	30	25	0	55	<.001
			% within Urine albumin - Pre	54.5%	45.5%	.0%	100.0%	
			% within Urine albumin - Day 2	51.7%	62.5%	.0%	55.0%	
		Severe	Count	0	11	2	13	<.001
			% within Urine albumin - Pre	.0%	84.6%	15.4%	100.0%	
			% within Urine albumin - Day 2	.0%	27.5%	100.0%	13.0%	
	Total		Count	58	40	2	100	
			% within Urine albumin - Pre	58.0%	40.0%	2.0%	100.0%	
			% within Urine albumin - Day 2	100.0%	100.0%	100.0%	100.0%	

* There is a significant decrease in urine albumin in both the groups.

TABULAR COLUMN 15-DURATION OF STAY IN CCU/LABOUR WARD

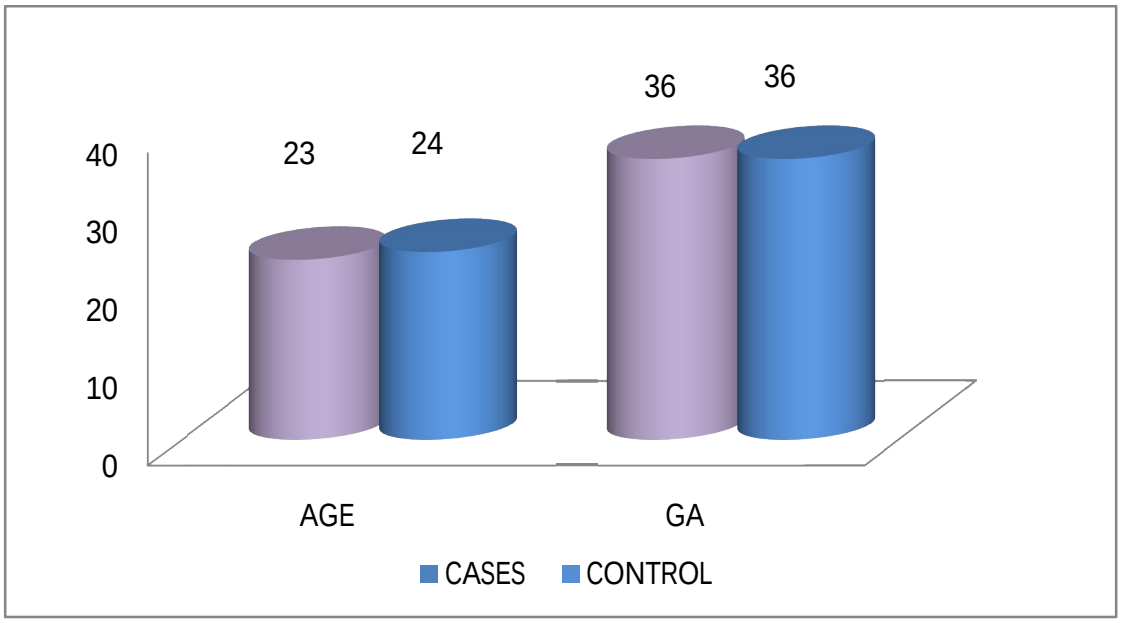
			Group		Total	P
			Control	Cases		
Duration of stay	1	Count	0	6	6	
		% within Duration of stay	.0%	100.0%	100.0%	
		% within Group	.0%	6.0%	3.0%	
	2	Count	52	94	146	
		% within Duration of stay	35.6%	64.4%	100.0%	
		% within Group	52.0%	94.0%	73.0%	
	3	Count	39	0	39	
		% within Duration of stay	100.0%	.0%	100.0%	<.001
		% within Group	39.0%	.0%	19.5%	
	4	Count	9	0	9	
		% within Duration of stay	100.0%	.0%	100.0%	
		% within Group	9.0%	.0%	4.5%	
Total		Count	100	100	200	
		% within Duration of stay	50.0%	50.0%	100.0%	
		% within Group	100.0%	100.0%	100.0%	

* There is a significant decrease (<.001) in the duration of stay in the study group due to rapid fall of BP and improvement of laboratory values.

DISCUSSION

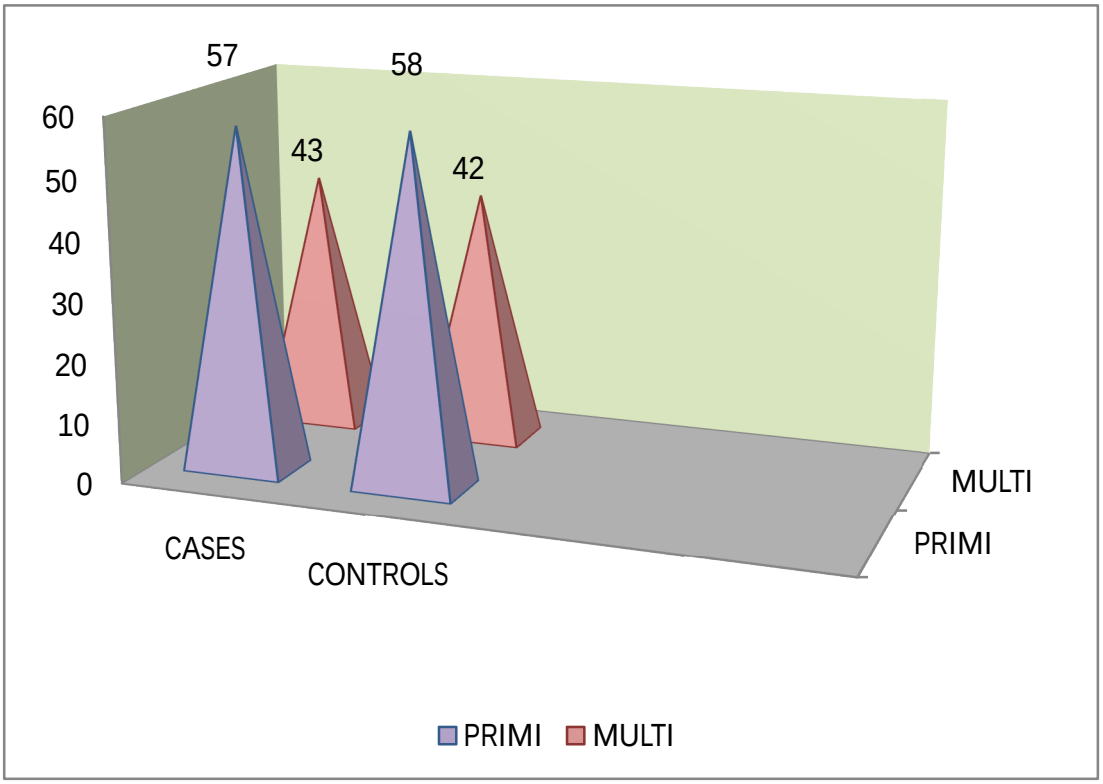
DISCUSSION

AGE AND GESTATIONAL AGE IN STUDY AND CONTROL GROUP



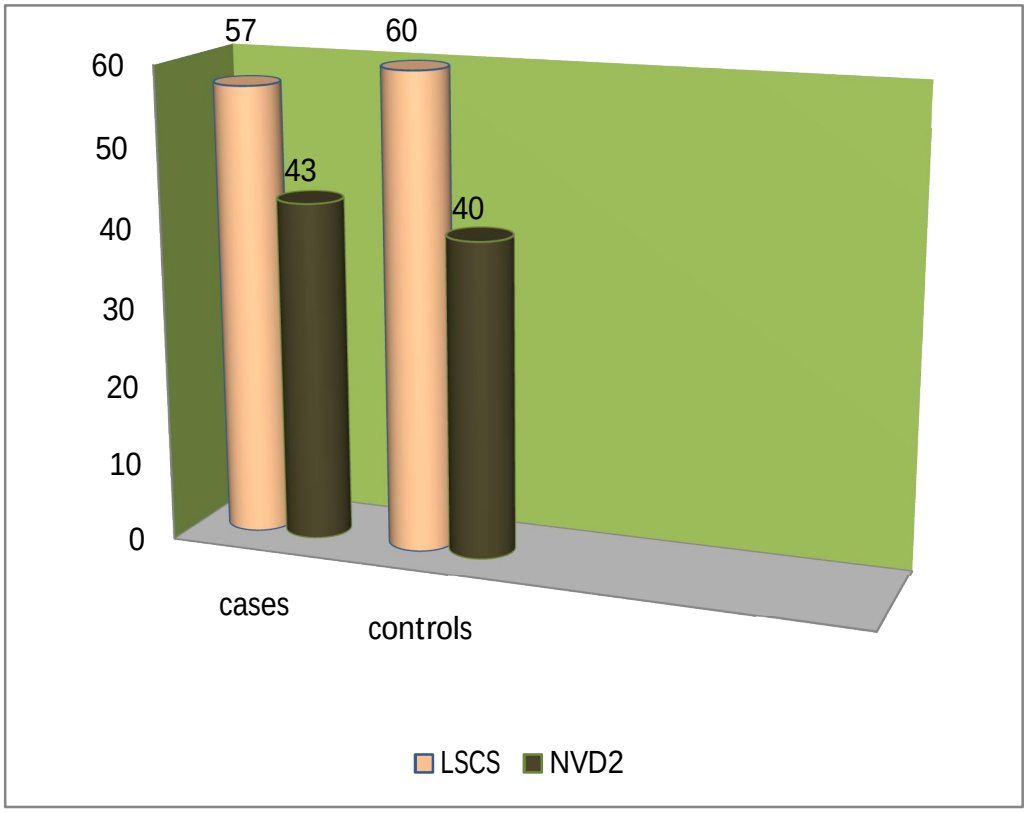
- ❖ The average age in both the study and control group was 23-24 years
- ❖ While the gestational age was 36-37 weeks in both the study and control group

PARITY



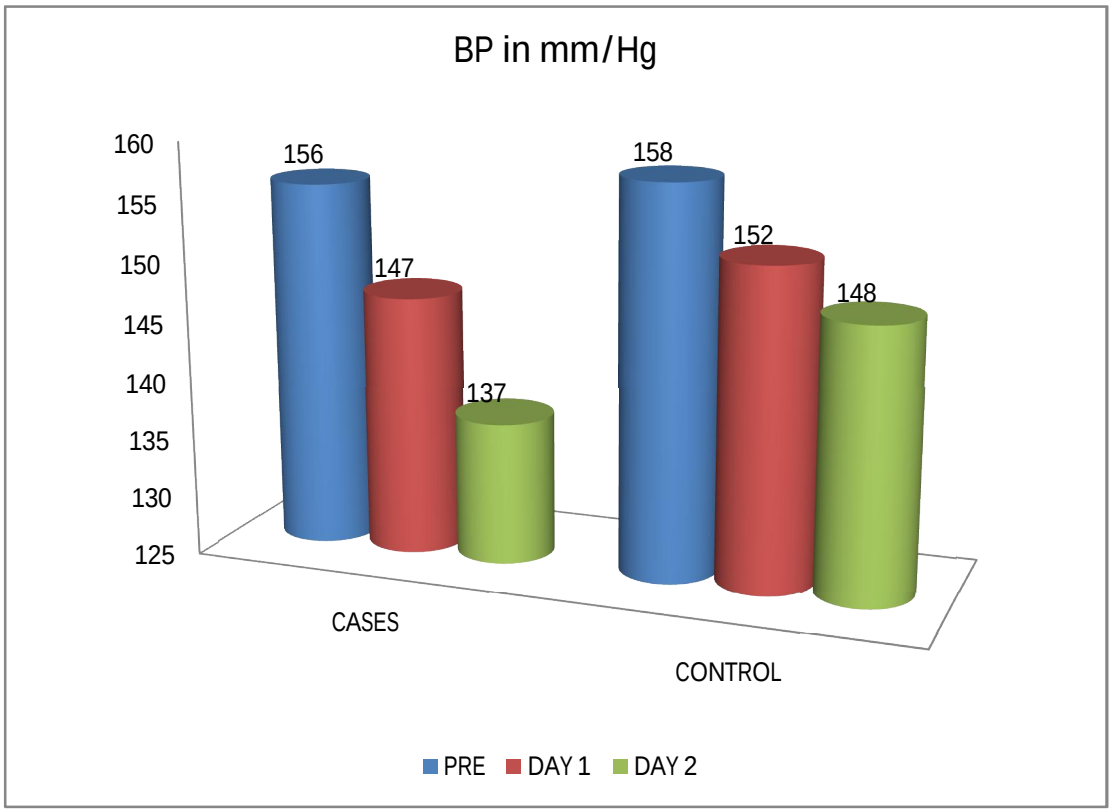
- Severe preeclampsia was most commonly seen in primigravida
- Cases-57% was primigravida
- Controls-58% was primigravida

MODE OF TERMINATION



❖ In this study 57% of cases and 60% of controls had LSCS while the remaining patients had a normal vaginal delivery(NVD).

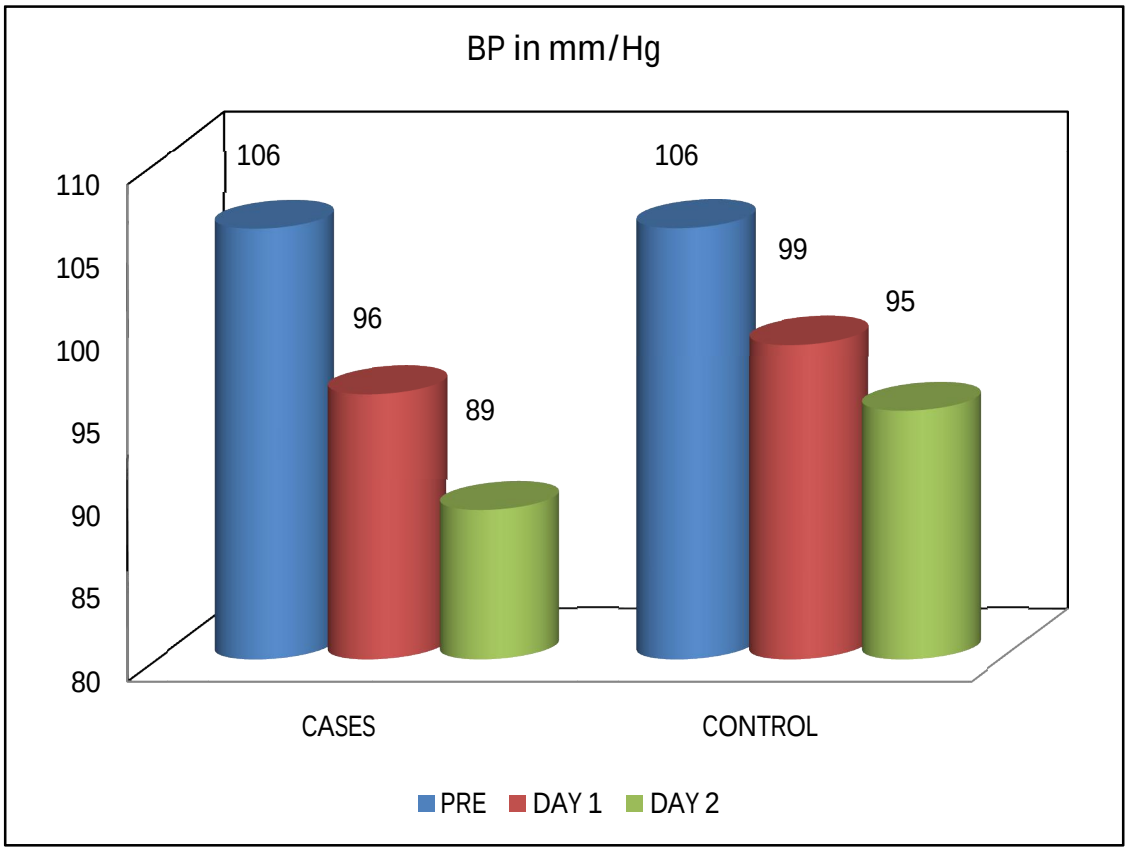
SYSTOLIC BLOOD PRESSURE



- The systolic blood pressure in the study group decreases from a mean value of 156mm/Hg to 147mm/Hg in the first postpartum day which further decreases to 137mm/Hg in the second postpartum day.

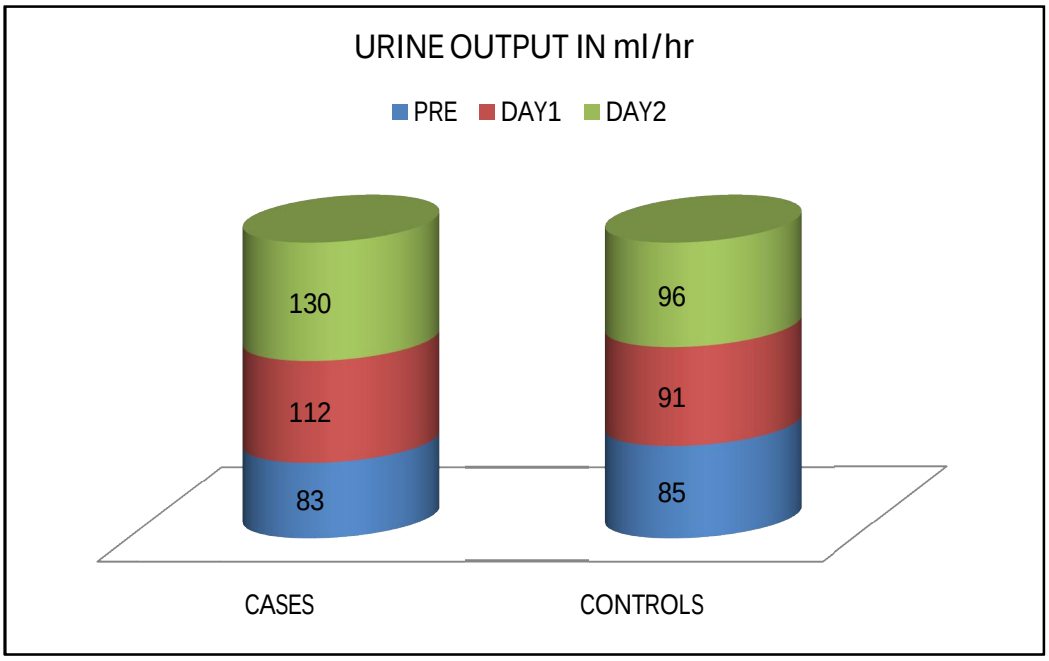
- The systolic blood pressure in the control group decreases from a mean value of 158mm/Hg to 152mm/Hg in the first postpartum day which further decreases to 148mm/Hg in the second postpartum day indicating a rapid recovery following curettage.

DIASTOLIC BLOODPRESSURE



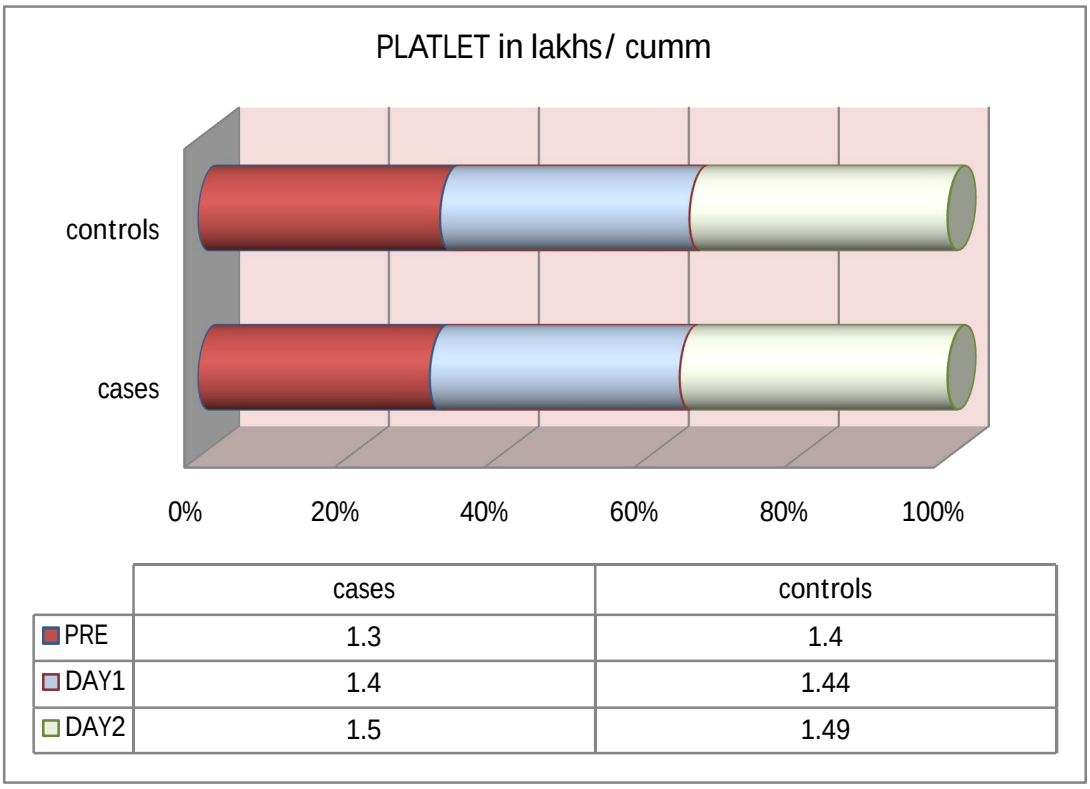
- The diastolic blood pressure in the study group decreases from a mean value of 106mm/Hg to 96mm/Hg in the first postpartum day which further decreases to 89mm/Hg in the second postpartum day.
- The diastolic blood pressure in the control group decreases from a mean value of 106mm/Hg to 99mm/Hg in the first postpartum day which further decreases to 95mm/Hg in the second postpartum day indicating a rapid recovery following curettage.

URINE OUTPUT



- The urine output in cases increased from a mean of 83ml/hr in the cases to about 130ml/hr in the second postpartum day when compared to the control group where the increase was just from a mean of 85ml/hr to 96ml/hr in the second postpartum day indicating an increase of about 25% in the study group when compared to the control group with a significant P value of <.001

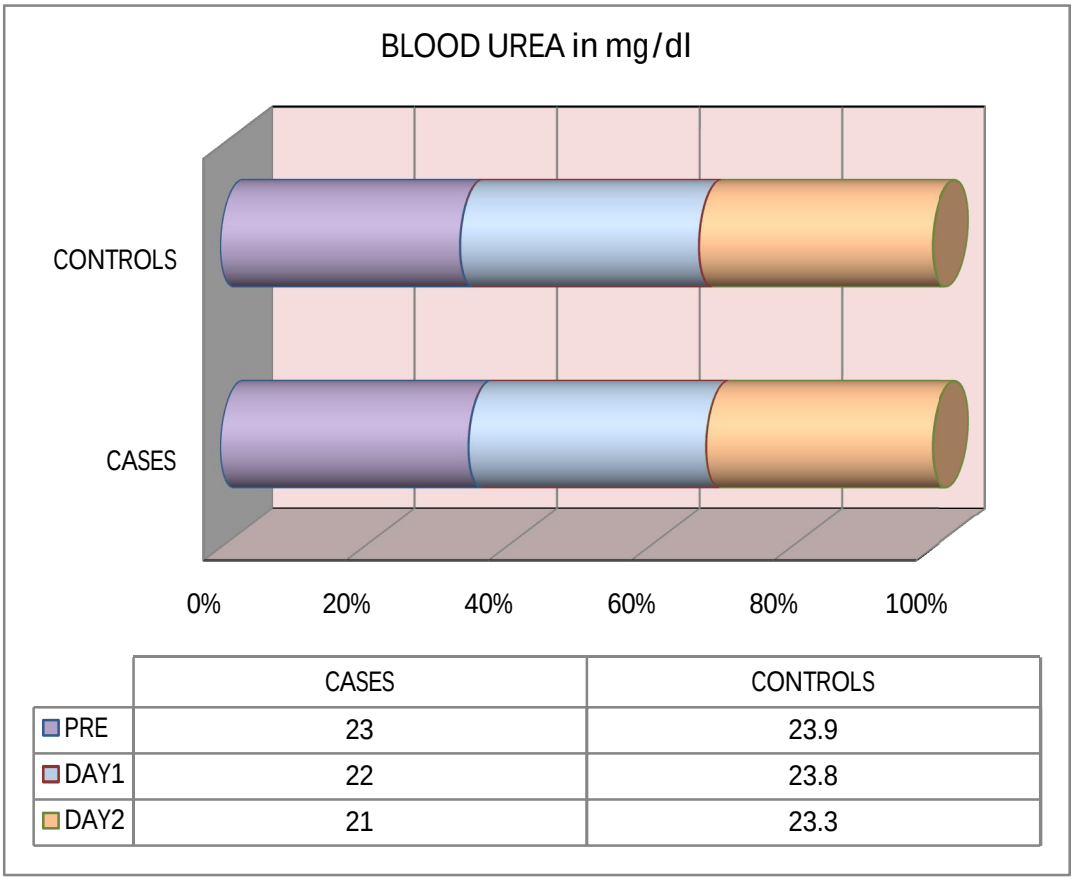
PLATELET COUNT



	cases	controls
PRE	1.3	1.4
DAY1	1.4	1.44
DAY2	1.5	1.49

- The platelet count increased in both the study and control group . The percentage of increase was found to be 13% in the study group and 4%in the control group indicating an increase of platelet count by 9% in the study group compared to the control group with a P value of .021 which is significant.

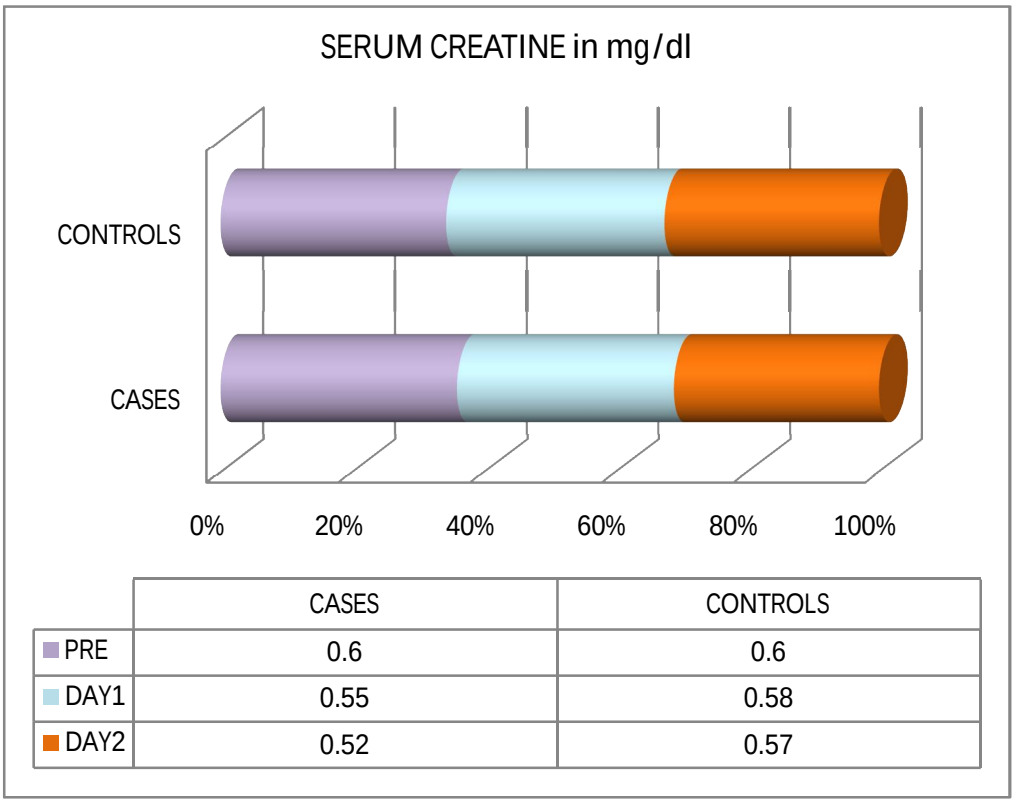
BLOOD UREA



	CASES	CONTROLS
PRE	23	23.9
DAY1	22	23.8
DAY2	21	23.3

- The decrease in blood urea was about 8% in the study group when compared to the control group which had a decrease of only 2% although all the patients in both the control and study group had blood urea levels within the normal limit with a P value of .002 which is significant.

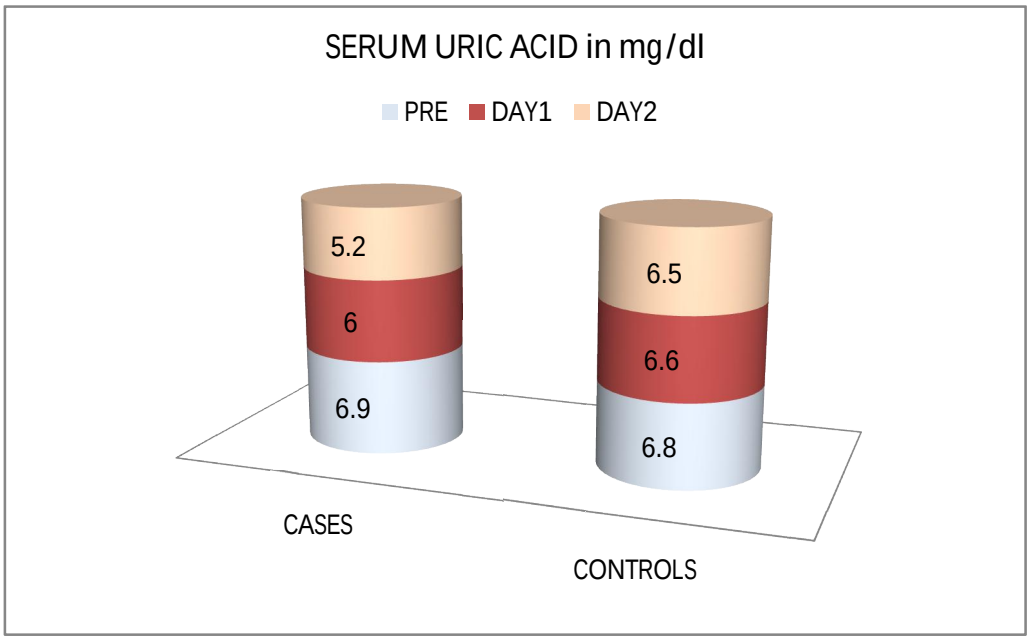
SERUM CREATININE



	CASES	CONTROLS
PRE	0.6	0.6
DAY1	0.55	0.58
DAY2	0.52	0.57

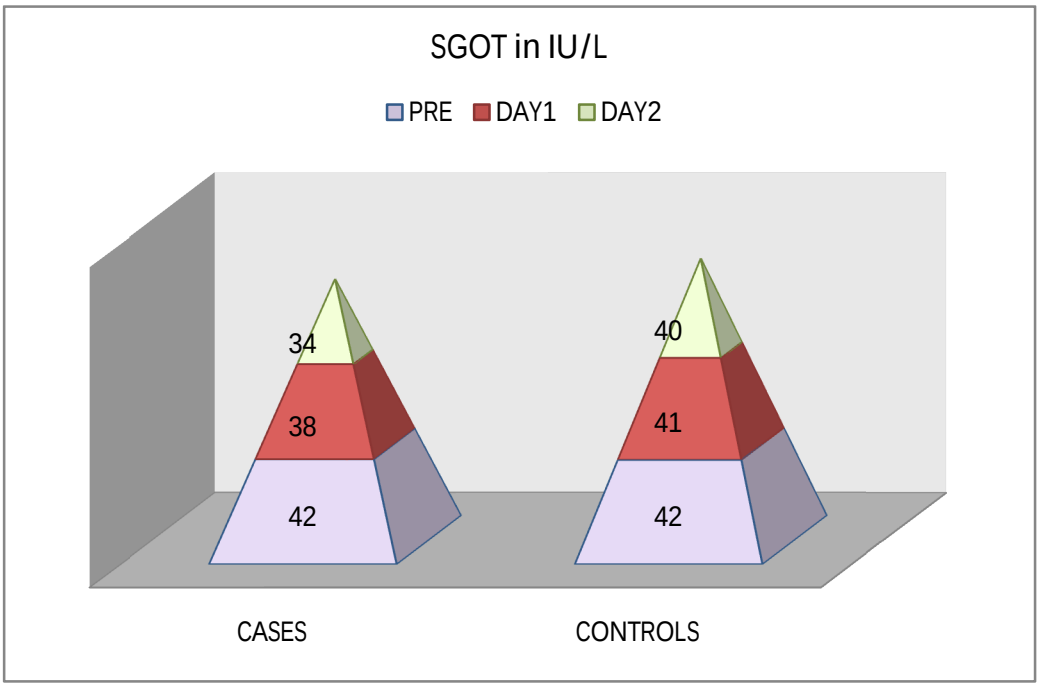
- The decrease in serum creatinine was about 13% in the study group when compared to the control group which had a decrease of only 5% although all the patients in both the control and study group levels had serum creatinine within the normal limit with a P value of .010 which is significant.

SERUM URIC ACID



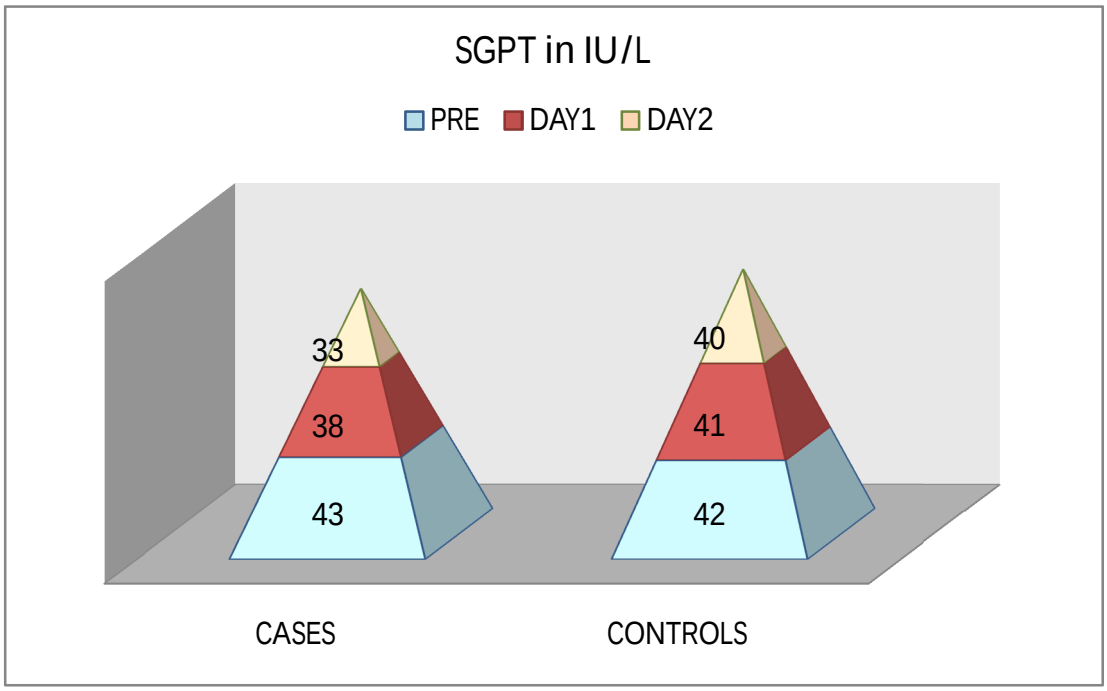
- The serum uric acid in cases decreased from a mean of 6.9mg/dl in the cases to about5.2mg/dl in the second postpartum day when compared to the control group where the decrease was just from a mean of 6.8mg/dl to 6.5mg/dl in the second postpartum day indicating a decrease of about 20% in the study group when compared to the control group with a significant P value of <.001.

SGOT



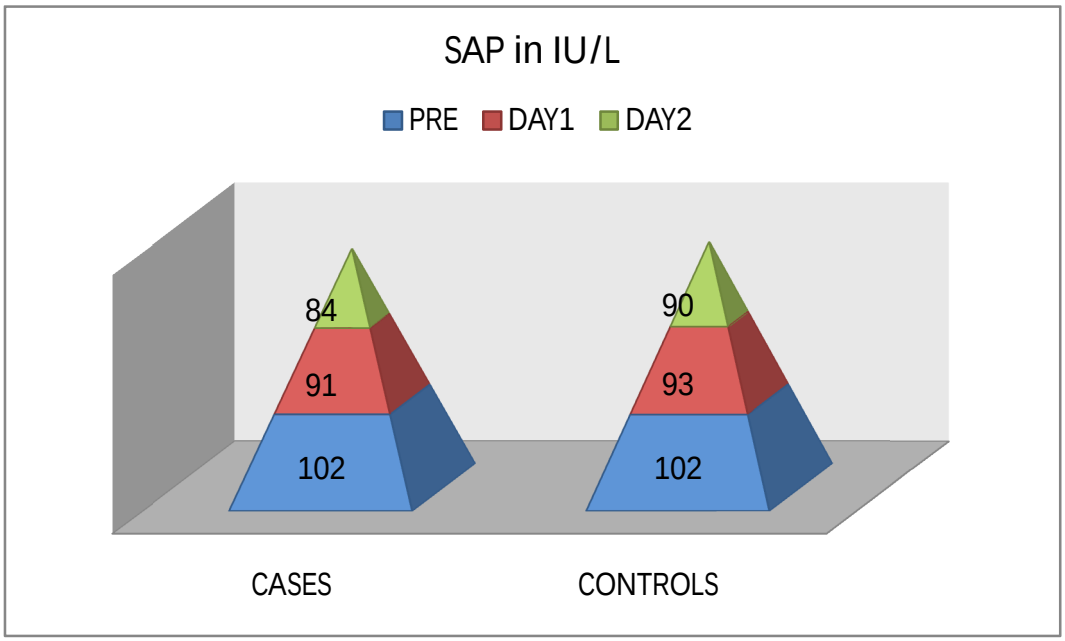
- The decrease in the SGOT value in the study group was about 19% when compared to the control group which had a decrease of only 2% with a P value of <.001 which is highly significant.

SGPT



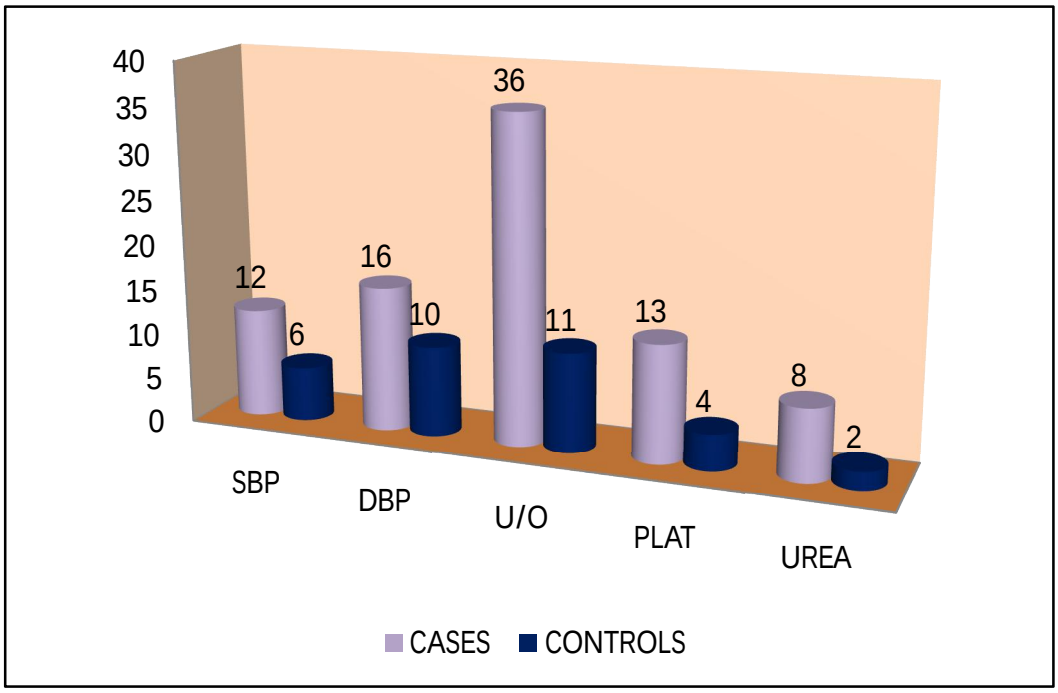
- The decrease in the SGPT value in the study group was about 23% when compared to the control group which had a decrease of only 4% with a P value of <.001 which is highly significant.

SAP



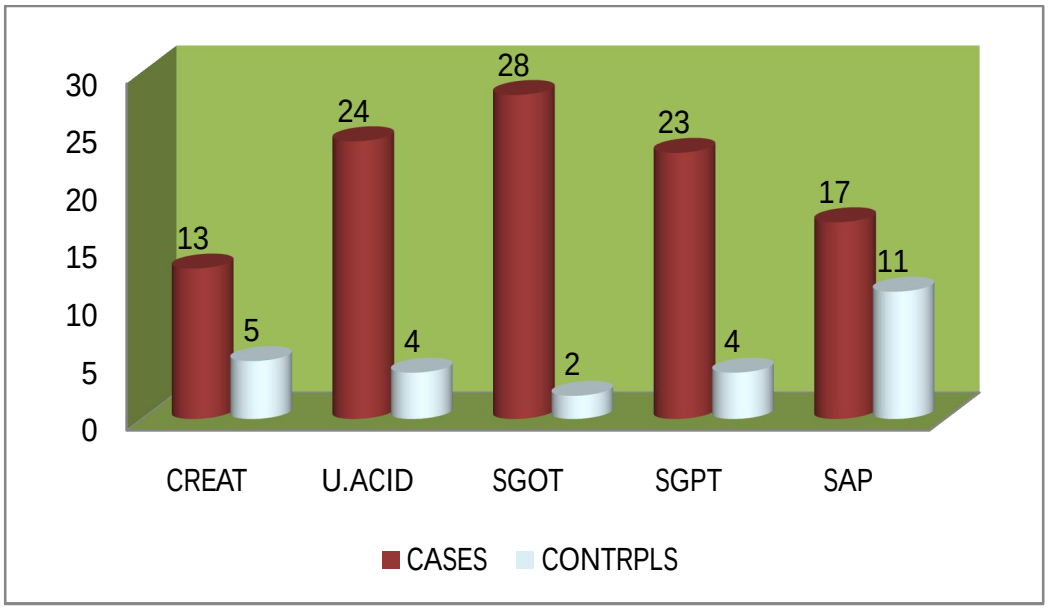
- The decrease in the SAP value in the study group was about 18% when compared to the control group which had a decrease of only 7% with a P value of <.001 which is highly significant

PERCENTAGE OF DIFFERENCE OF THE VALUES MONITORED ON ADMISSION AND ON DAY2 IN BOTH CONTROL AND CASES



- From this study it is seen that there is a significant difference between the cases and controls as follows
- Systolic blood pressure(SBP) decreased by 12%in the study group when compared to the control group which had a decrease of only 6% with a p value of <.001
- Diastolic blood pressure(DBP) decreased by 16% in the study group compared to the control group with a decrease of about 10%
- Urine output(U/O) increased by 36% in the study group while the control group had an increase of 11% with a p value <.001.

- Platelet count(PLAT) had a increase of 13% in the study group and 4% in the control group with a p value of .021
- Blood urea had a decrease of 8% in the study group and 2% in the control group with a significant p value of.002



- Creatinine (CREAT) had a decrease of 13% in the study group with only 5% decrease in the in the control group with a p value of .010
- Serum uric acid (U.ACID) had a decrease of 24% in study group and only 4% decrease in control group with a p value of <.001.
- Liver enzymes also had a significant decrease in the study group when compared to the control group with a p value of <.001

SGOT : cases -decreased by 28%

Controls –decreased by 2%

SGPT : cases -decreased by 23%

Controls –decreased by 4%

SAP : cases -decreased by 17%

Controls –decreased by 11%

CONCLUSION

CONCLUSION

Preeclampsia is the one of the leading cause of maternal death worldwide. It constitutes about 22% of all perinatal deaths and about 30% of maternal deaths in developed countries. Intracranial bleeding is the leading cause of maternal death in preeclampsia.

The morbidity of severe preeclampsia can be prevented by early treatment and accelerated recovery from preeclampsia.

This study states that postpartum curettage is a safe and effective procedure which accelerates recovery from preeclampsia averting complications and decreasing the mortality and morbidity associated with severe preeclampsia.

ANNEXURE

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CONSENT FORMS

INFORMED CONSENT FORM

Hypertensive disorders during pregnancy continue to be a major cause of maternal and perinatal morbidity and mortality worldwide .In developing countries they are second only to anaemia with approximately 3-10% of all pregnancies.

Preeclampsia resolves only after all gestational products particularly the placenta and decidua have been removed or have ceased to function. Therefore removal is the best means of cure of the disease. We consider the above theory and therefore by immediate post partum curettage we try to remove all the decidual tissues therefore hastening the recovery. This study is to observe the effects of immediate post partum curettage on resolution of clinical and laboratory indices associated with severe PIH and eclampsia .The identity of the patients will be kept confidential**.**

INFORMED CONSENT FORM

STUDY TITLE : ".ABILITY OF POSTPARTUM CURETTAGE TO ACCELERATE MATERNAL RECOVERY IN SEVERE PREECLAMPSIA PATIENTS"

STUDY CENTRE : RSRM Lying in hospital , Royapuram,Chennai

Patient may check (✓) these boxes.

Participant Name : Age: I.P.no:

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without my legal rights being affected. ☐

I understand that investigator, the institution, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study.

☐

I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I hereby consent to undergo complete physical examination diagnostic tests including hematological, biochemical, radiological and urine examinations and intervention surgical procedure like curettage.

☐

I hereby consent to participate in this study of "ABILITY OF POSTPARTUM CURETTAGE TO ACCELERATE MATERNAL RECOVERY IN SEVERE PREECLAMPSIA PATIENTS."

☐

Signature of patient /gaurdian: .....

Place .....Date…….....

Address

…….……

……...

Signature of investigator.....

place:…….date……..

PROFORMA

PROFORMA

S.No: Unit:

NAME: AGE: I.P.No:

OCCUPATION: INCOME:

SOCIO ECONOMIC STATUS:

RESIDENCE:

PRESENTING HISTORY :-

OBSTETRIC INDEX LMP: EDD

pedal edema, pain abdomen, bleeding / draining per vaginum, perception of fetal movements , epigastric pain ,headache, nausea, vomiting ,decreased urine output, blurring of vision

MENSTRUAL HISTORY Menarche: Regular /Irreg.

LMP: EDD:

MARITAL HISTORY

OBSTETRIC HISTORY

LCB

Previous Obstetric Performance

1. History of IUD
2. History of IUGR
3. H/o.PIH in the previous pregnancy
4. H/O miscarriages

OTHER COMPLICATIONS ASSOCIATED

1. Anaemia
2. Heart disease
3. Gestational diabetes mellitus
4. RH negative pregnancy
5. IUGR

CLINICAL EXAMINATION

Ht : Wt:

Anaemia

Edema Legs

PR: CVS:

BP: RS:

ABDOMINAL EXAMINATION

Uterine size in gestatational weeks

FH

LIQUOR CLINICALLY

PV

INVESTIGATIONS

Urine :

Alb:

Sugar and deposits:

24 our urinary protein

Blood :

Hb %:	LFT	RFT
PCV	Srbilurubin	Urea
Platelets	total protein	Srcreat
	Sr albumin	
BT/CT	SGOT	
Gr/ Typing:	SGPT	
	SAP	

ULTRASONOGRAM

BPD

FL:

HC:

AC: LIQUOR:

PLACENTA:

MODE OF DELIVERY: CAESAREAN / VAGINAL DELIVERY

POST PARTUM CURETTAGE: DONE / NOT DONE

ANTIHYPERTENSIVES GIVEN

MONITORING PATIENTS IN POSTPARTUM PERIOD

BP MONITORING EVERY 4th HOURLY

DAY1 BP						
DAY2 BP						

MONITORING URINE OUTPUT AND URINE ALBUMIN 4th HOURLY

DAY1	U/O						
	U/A						
DAY2	U/O						
	U/A						

MONITORING LABORATORY VALUES DAILY

	PLAT	**UREA**	**CREAT**	**URICACID**
Day1				
Day2				

	SGOT	**SGPT**	**SAP**
Day1			
Day2			

DURATION OF STAY IN CCU/LABOUR WARD:

ABBREVATIONS

ABBREVIATIONS

NK	:	Natural killer
HLA-G	:	Human leucocyte antigen G
sFlt	:	soluble Fms like tyrosine kinase
sEng	:	soluble endoglin
ROS	:	Reactive oxygen species
AGEs	:	Activated Glycogen end products
NO	:	Nitric oxide
PlGF	:	Placental growth factor
VEGF	:	Vascular endothelial growth factor
TGF	:	Transforming growth factor
NICE	:	National Institute of Clinical Excellence
PCV	:	Packed cell volume
Beta HCG	:	beta Human chorionic gonadotrophin
PAPP-A	:	Pregnancy associated plasma protein
CLASP	:	Collaborative low dose aspirin study
TGL	:	Triglyceride
LDL	:	Low density lipoprotein
BMI	:	Body mass index
ESRD	:	End stage renal disease
IUGR	:	Intrauterine growth restriction

SGOT(AST)	:	Serum glutamic oxaloacetic transaminase (Aspartate transaminase)
SGPT (ALT)	:	Serum glutamic pyruvic transaminase (Alanine transaminase)
SAP	:	Serum alkaline phosphatase
LDH	:	Lactate dehydrogenase
CTG	:	Cardiotocogram
APLA	:	Antiphospholipid antibody syndrome
CCU	:	Critical care unit
NS	:	Not significant

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Ability of postpartum curettage to accelerate maternal Recovery in severe preeclampsia patients

Principal Investigator : Dr.K.Karthiga

Designation : PG in MS(OG)

Department : Department of OG
Government Stanley Medical College,
Chennai-1

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 08.07.2013 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

12/8/13

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MASTER CHART

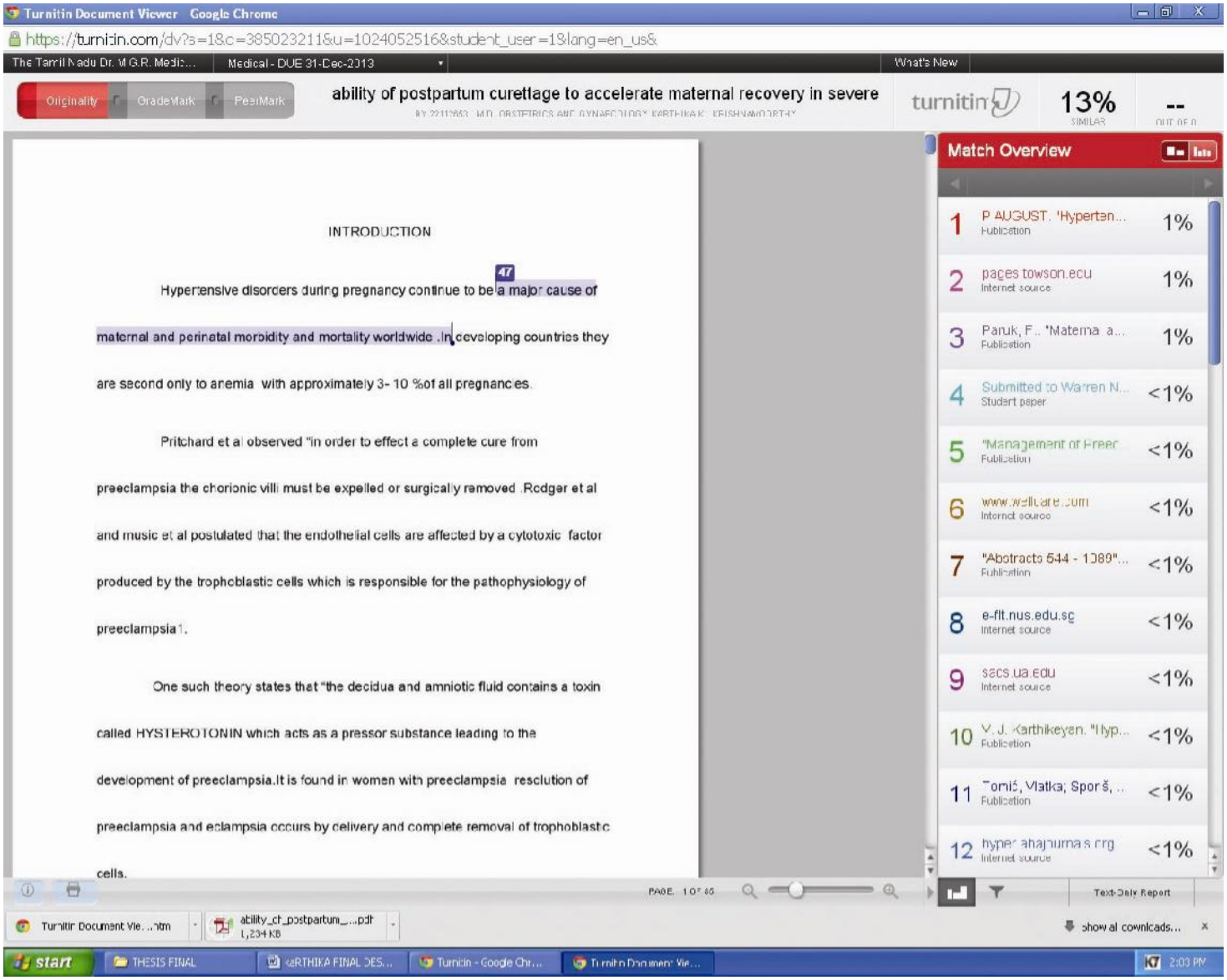
S.No	Age	Parity	Gestational age(wks)	CASES - CURETTAGE DONE PATIENTS																DAY 1														DAY 2														DURATION OF STAY IN CCU/LA
				Imminent symptoms	systolic BP(mmHg)	diastolic BP(mmHg)	Urine albumin	Urine output	Platelet count (lak/hs/litre	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	Labetalol	Nifidepine	MgSO4	Mode of termination	systolic BP mm/Hg	diastolicBP mm/Hg	Urine albumin	Urine output (ml/hr)	Platelet count lak/lit	Bl.Ureamg/dl	S.Creatininemg/dl	Uric acidmg/dl	SGOTmeq/lt	SGPTmeq/lt	SAPmeq/lt	Labetalol	Nifidepine	MgSO4	systolicBPmm/Hg	diastolicBPmm/Hg	Urine albumin	Urine output(ml/hr)	Platelet countlakh	Bl.Ureamg/dl	S.Creatininemg/dl	Uric acidmg/dl	SGOTmeq/li	SGPTmeq/lt	SAPmeq/lt	Labetalol	Nifidepine	MgSO4	
1	23	Primi	37		160	110	1	100	1	24	0.8	7.8	32	36	97	2	2		LSCS	150	100	1	100	1.5	24	0.8	6.2	32	36	96	2	2		140	90	trace	150	1.5	23	0.7	5.9	32	36	95	2			2
2	28	Primi	37		160	110	2	50	1.2	22	0.7	7.2	34	30	95	2	2		LSCS	150	90	1	100	1.2	21	0.7	6	32	30	95	2	1		140	90	1	100	1.3	20	0.7	4.8	32	30	95	2			2
3	27	Primi	38		150	100	2	100	1.4	26	0.6	6.3	28	25	87	2	2		LSCS	140	90	1	150	1.5	26	0.6	4.2	28	25	86	2			130	90	trace	150	1.5	25	0.6	4	28	25	86	2			1
4	20	Primi	30	**headache, decreased urine output**	170	120	3	30	1	30	0.9	9.2	72	78	186	2	2	+	NVD	150	100	2	50	1.4	28	0.8	5.8	69	63	150	2	2	+	140	90	1	100	1.7	23	0.7	4.1	42	40	97	2	1		2
5	20	G3P2L2	35	**headache**	160	120	2	50	1.2	23	0.8	6.8	99	99	192	1	1	+	LSCS	160	90	trace	100	2	23	0.8	5	83	78	137	2	1	+	130	90	trace	150	2.1	22	0.6	4	45	48	94	2			2
6	21	Primi	37		150	110	2	100	1.8	18	0.5	6	43	35	75	1	1		LSCS	140	100	1	100	1.8	18	0.5	4.9	43	35	74	2	1		130	90	trace	100	1.8	16	0.4	4.7	42	35	74	2			2
7	22	Primi	37		150	100	3	150	1.5	18	0.5	5.9	34	38	66	2	2		LSCS	140	90	2	150	1.5	18	0.5	5.8	33	37	90	2			130	80	1	200	1.6	18	0.4	4.9	33	37	90	1			2
8	20	Primi	38		170	110	2	100	1.6	26	0.6	6.8	39	37	91	2	2	+	LSCS	150	100	1	150	1.6	25	0.6	6.6	39	37	90	2	2	+	140	90	trace	150	1.7	26	0.6	5	39	37	90	2			2
9	19	Primi	35		160	100	1	100	1.5	18	0.5	7.2	31	35	91	2	2		NVD	150	100	1	100	1.5	18	0.5	7.1	30	35	91	2	2		150	90	1	150	1.5	16	0.5	6.1	30	35	91	2	1		2
10	20	Primi	36		150	110	2	100	1.8	20	0.5	7.9	32	34	82	2	2		NVD	140	100	1	100	1.8	20	0.5	6.8	32	34	80	2	1		140	90	1	100	2	18	0.5	5.9	32	34	80	2			2
11	26	G3P1L1D1	37		150	100	2	50	1.4	22	0.4	7	49	45	98	2	2		LSCS	140	90	2	100	1.6	22	0.3	6	44	43	95	2			130	90	1	150	1.6	21	0.4	5.1	42	42	94	2			1
12	28	G2P1L1`	37		150	100	**2**	50	1.5	24	0.6	6.8	36	38	87	2	2		NVD	140	100	2	100	1.5	24	0.5	5.9	34	36	87	2	1		140	90	1	100	1.5	24	0.5	5.7	34	35	86	2			2
13	25	G2P1L1	36		160	100	2	100	1.7	26	0.5	6.4	34	39	89	2	2		NVD	140	90	1	150	1.6	25	0.5	6	34	36	88	2			130	90	trace	150	1.7	25	0.5	5.4	32	34	87	2			2
14	24	Primi	37		60	100	2	100	1.9	28	0.7	6.2	22	28	89	2	2		LSCS	150	100	1	100	1.9	28	0.7	5.8	22	27	87	2	2		140	90	trace	100	2	28	0.7	5	22	27	87	2	1		2
15	23	Primi	37		160	100	2	100	1.6	20	0.6	6.6	35	30	88	2	2		LSCS	150	100	2	100	1.6	20	0.6	6	35	29	87	2	2		130	90	1	150	1.6	18	0.5	5.1	35	28	86	2	1		2
16	20	Primi	38		150	100	2	100	1.5	18	0.5	6.1	34	31	96	2	2		LSCS	140	100	1	150	1.5	18	0.5	5.4	34	31	90	2	1		130	90	1	150	1.6	17	0.5	5	34	31	90	2			2
17	20	Primi	37	**headache, decreased urine output**	150	110	3	30	1	32	0.9	8.8	72	69	158	1	1	+	LSCS	140	100	2	100	1.3	26	0.8	6.6	43	40	98	2	1	+	130	90	1	150	1.5	25	0.6	5.3	40	39	96	2			2
18	21	Primi	37		160	110	1	100	1.5	28	0.5	7	36	31	85	1	1		LSCS	150	90	1	100	1.5	24	0.5	6.2	35	31	80	2	1		140	90	trace	150	1.6	22	0.4	5.9	34	30	80	2			2
19	21	Primi	36		150	100	2	50	1.4	26	0.4	6.8	38	32	97	1	1		LSCS	140	100	1	100	1.3	24	0.3	6.1	35	32	95	2	1		140	90	trace	100	1.5	20	0.3	5.8	35	32	94	2			2
20	22	G2P1L1	37		160	100	1	100	1.3	25	0.6	6.1	39	35	87	2	2		LSCS	150	90	trace	150	1.4	25	0.6	5.7	37	35	86	2	1		140	80	trace	150	1.4	25	0.5	5.7	37	34	84	2			2
21	20	Primi	37		160	100	1	100	1.2	24	0.5	6.6	37	29	78	2	2		LSCS	150	90	1	150	1.2	24	0.5	6.5	37	29	77	2	1		140	90	trace	150	1.2	24	0.5	6	37	29	77	2			2
22	21	Primi	38		150	100	1	50	1.6	25	0.4	5.9	29	23	99	1	1		LSCS	140	100	1	100	1.6	25	0.4	5.8	28	21	78	2	2		130	90	trace	100	1.6	20	0.4	5	28	21	78	2			2
23	24	G2P1L1	32	**headache ,decreased urine output**	170	100	3	30	1	30	0.9	9.7	101	143	200	2	2	+	NVD	150	100	2	50	1.6	26	0.6	8.3	72	101	143	2	1	+	140	90	2	100	1.8	23	0.5	6.2	46	52	98	2	1		2
24	23	G2P1L1	40		160	100	2	100	1.5	20	0.4	7.1	24	29	67	2	2		NVD	150	90	1	150	1.7	20	0.4	6.9	24	29	65	2	2		140	90	1	150	1.7	20	0.4	6.3	24	29	65	2			2
25	22	Primi	35	**headache, blurring of vision**	160	110	2	100	1	28	0.6	7.2	76	78	165	2	2	+	LSCS	150	100	1	100	1.3	26	0.6	6.5	45	50	107	2	1	+	140	90	1	150	1.6	25	0.6	5.9	40	40	99	2	1		2
26	23	Primi	37		160	100	1	100	1.6	18	0.5	6.8	29	23	71	1	1		LSCS	160	90	1	100	1.6	18	0.5	6.7	29	23	70	2	1		150	80	trace	100	1.6	18	0.4	6.3	29	23	70	2			2
27	20	Primi	36		160	110	1	50	1.4	20	0.4	6.6	23	20	68	2	2		LSCS	140	100	1	50	1.4	20	0.4	6	20	20	68	2	1		130	90	trace	100	1.4	20	0.3	5.8	20	19	67	2			1
28	21	G3P1L1A1	37		150	110	2	100	1.2	18	0.6	5.9	35	37	95	2	2		LSCS	140	100	1	150	1.2	18	0.6	5.7	35	36	94	2	1		130	90	trace	150	1.4	16	0.5	5.5	35	36	93	2			1
29	19	Primi	34		150	110	2	100	1.4	22	0.7	6.6	29	21	78	2	2		NVD	140	100	1	100	1.4	22	0.7	5.8	28	20	78	2	1		130	90	1	150	1.6	20	0.6	4.9	25	20	76	2			2
30	18	Primi	37		160	110	1	100	1.6	24	0.5	6.9	30	35	91	2	2		LSCS	140	100	trace	100	1.6	22	0.5	5	30	35	90	2	1		140	90	trace	100	1.8	22	0.5	4.6	30	35	90	2			2
31	20	G2P1L1	37		160	110	1	100	1.2	24	0.7	7.6	32	38	96	2	1		NVD	160	100	1	100	1.2	24	0.7	6.9	32	38	95	2	1		140	90	trace	150	1.3	22	0.6	5.1	32	38	95	2			2
32	24	G2P1L1	36		150	100	2	50	1.3	26	0.6	6.3	26	25	85	2	1		NVD	140	90	1	100	1.3	25	0.6	5.9	26	25	85	1			130	90	1	100	1.3	25	0.6	4.2	26	25	85	2			2
33	25	G2P1L1	38		160	120	2	100	1.1	25	0.6	6	28	27	98	2	2	+	NVD	150	100	1	150	1.1	25	0.5	5.1	28	27	96	2	1	+	140	90	trace	150	1.2	25	0.5	4.7	28	27	95	2			2
34	18	Primi	38	**headache,decreased urine output,**	170	110	3	30	1	35	0.9	8.8	72	71	183	2	2	+	LSCS	150	100	2	50	1.3	28	0.7	7.9	55	51	155	2	1	+	140	90	1	100	1.5	26	0.7	6	43	39	82	2			2
35	20	Primi	34		150	110	2	100	1.5	21	0.5	6.2	31	39	73	2	1		NVD	140	90	1	100	1.4	20	0.5	5.5	31	35	70	1			130	80	trace	100	1.2	20	0.9	5	31	35	70	2			2
36	21	Primi	37		160	110	1	50	1.4	23	0.3	6.3	29	29	74	2	1		LSCS	150	100	1	100	1.4	22	0.3	5.1	29	28	74	2	1		140	90	trace	100	1.5	21	0.3	4.8	29	28	74	2			2
37	23	Primi	38		150	110	1	100	1.6	18	0.7	5.9	37	35	95	2	1		LSCS	140	100	trace	100	1.6	18	0.6	5	37	34	94	2	1		140	90	trace	100	1.7	18	0.6	4.5	37	34	94	2			2
38	26	G3P1L1A1	36		160	110	1	150	1.5	20	0.6	6.8	30	38	93	1	1		LSCS	150	100	1	150	1.5	20	0.5	6.5	30	36	93	2	1		140	90	1	200	1.6	19	0.5	4.9	30	36	93	2			2
39	22	Primi	35		160	100	2	150	1.4	20	0.6	6.5	30	34	96	1	1		NVD	160	90	1	150	1.4	20	0.6	6	30	34	95	2	1		140	90	trace	150	1.5	20	0.5	5	30	32	92	2			2

S.No	Age	Parity	Gestational age(wks)	CASES - CURETTAGE DONE PATIENTS																DAY 1														DAY 2														DURATION OF STAY IN CCU/LA
				Imminent symptoms	systolic BP(mmHg)	diastolic BP(mmHg)	Urine albumin	Urine output	Platelet count (lak/hs/litre	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	Labetalol	Nifidepine	MgSO4	Mode of termination	systolic BP mm/Hg	diastolicBP mm/Hg	Urine albumin	Urine output (ml/hr)	Platelet count lak/lit	Bl.Ureamg/dl	S.Creatininemg/dl	Uric acidmg/dl	SGOTmeq/lt	SGPTmeq/lt	SAPmeq/lt	Labetalol	Nifidepine	MgSO4	systolicBPmm/Hg	diastolicBPmm/Hg	Urine albumin	Urine output(ml/hr)	Platelet countlakh	Bl.Ureamg/dl	S.Creatininemg/dl	Uric acidmg/dl	SGOTmeq/li	SGPTmeq/lt	SAPmeq/lt	Labetalol	Nifidepine	MgSO4	
40	21	Primi	34	**headache**	150	110	2	100	1.8	18	0.5	5.9	35	39	71	1	1	+	NVD	140	100	1	100	1.9	18	0.5	5	34	36	69	2	1	+	130	90	trace	150	1.9	18	0.5	4.5	34	36	94	2			2
41	20	primi	37	**epigastric pain ,decreased urine output**	160	120	3	30	1	36	0.9	8.9	101	107	206	2	1	+	LSCS	150	100	2	150	1.5	30	0.7	7.2	88	86	154	2	1	+	140	90	1	100	1.6	26	0.7	6.1	46	40	96	2			2
42	19	Primi	38		170	100	2	100	1.2	22	0.4	5.8	28	25	78	2	1	+	LSCS	150	90	1	100	1.2	21	0.4	5	28	25	76	2	1	+	140	90	1	100	1.4	21	0.3	4.8	28	25	76	2			2
43	25	G2P1L1	37		160	100	2	100	1.3	24	0.7	5.7	37	39	76	2	1		LSCS	150	90	1	150	1.4	23	0.6	4.9	35	37	75	2	1		140	90	trace	150	1.4	22	0.6	4.3	35	37	75	2			2
44	24	G2P1L1	30	**epigastric pain ,blurring of vision**	160	110	1	50	1.5	17	0.6	6.8	95	98	164	2	1	+	NVD	150	100	trace	100	1.5	17	0.5	6.5	72	78	111	2	1	+	140	90	trace	100	1.5	16	0.5	5.9	50	49	93	2			2
45	28	G3P2L2	37		150	110	2	100	1.9	16	0.3	6.5	24	25	66	2	1		LSCS	150	100	1	100	1.9	15	0.3	6	24	24	65	2	1		140	90	trace	150	1.4	15	0.2	5.3	24	24	65	2			2
46	29	G2P1L1	32		160	100	2	100	1.8	16	0.4	6.4	37	39	72	2	1		NVD	140	100	1	150	1.8	16	0.4	5.9	35	37	72	2	1		130	90	trace	150	2	15	0.4	4	35	37	72	2			2
47	20	Primi	37	**headache ,decreased urine output**	170	100	2	40	1	35	0.8	8.7	39	40	86	2	1	+	LSCS	160	90	1	100	1.3	30	0.7	7.9	38	40	80	2	1	+	140	90	1	100	1.5	26	0.7	6.1	35	39	80	2			2
48	19	Primi	36	**headache ,decreased urine output**	160	110	2	50	1	33	0.8	8.5	61	59	164	2	1	+	NVD	150	90	1	100	1.2	29	0.6	7.8	56	56	103	2	1	+	140	90	1	100	1.4	25	0.6	6	42	40	80	2			2
49	27	Primi	37		160	100	1	100	1.7	20	0.7	6.6	31	33	74	2	1		LSCS	160	90	trace	150	1.7	20	0.6	6.6	31	32	72	2	1		140	90	trace	150	1.7	20	0.5	5.3	31	30	70	2			2
50	25	Primi	38		160	110	1	100	1.6	21	0.7	6.5	22	26	66	2	1		LSCS	150	100	trace	100	1.6	21	0.6	5.5	22	26	66	2	1		140	90	trace	150	1.6	19	0.6	4.8	22	25	64	2			2
51	20	Primi	34	**decreased urine output,**	150	110	3	30	1	32	0.9	8.8	72	69	158	1	1	+	NVD	140	100	2	100	1.3	26	0.8	6.6	43	40	98	2	1	+	130	90	1	150	1.5	25	0.6	5.3	40	39	96	2			2
52	21	G2A1	36		160	110	1	100	1.5	28	0.5	7	36	31	85	1	1		LSCS	150	90	1	100	1.5	24	0.5	6.2	35	31	80	2	1		140	90	trace	150	1.6	22	0.4	5.9	34	30	80	2			2
53	24	G3A2	38		150	100	2	50	1.4	26	0.4	6.8	38	32	97	1	1		LSCS	140	100	1	100	1.3	24	0.3	6.1	35	32	95	2	1		140	90	trace	100	1.5	20	0.3	5.8	35	32	94	2			2
54	26	Primi	37		160	100	1	100	1.3	25	0.6	6.1	39	35	87	2	2		LSCS	150	90	trace	150	1.4	25	0.6	5.7	37	35	86	2	1		140	80	trace	150	1.4	25	0.5	5.7	37	34	84	2			2
55	23	Primi	38		160	100	1	100	1.2	24	0.5	6.6	37	29	78	2	2		LSCS	150	90	1	150	1.2	24	0.5	6.5	37	29	77	2	1		140	90	trace	150	1.2	24	0.5	6	37	29	77	2			2
56	19	Primi	34		150	110	1	50	1.6	25	0.4	5.9	29	23	99	1	1		NVD	140	100	1	100	1.6	25	0.4	5.8	28	21	78	2	2		130	90	trace	100	1.6	20	0.4	5	28	21	78	2			2
57	20	G2P1L0	30	**headache ,**	170	100	3	30	1	30	0.9	9.7	101	143	200	2	2	+	NVD	150	100	2	50	1.6	26	0.6	8.3	72	101	143	2	1	+	140	90	2	100	1.8	23	0.5	6.2	46	52	98	2	1		2
58	24	G2P1L1	34		160	100	2	100	1.5	20	0.4	7.1	24	29	67	2	2		NVD	150	90	1	150	1.7	20	0.4	6.9	24	29	65	2	2		140	90	1	150	1.7	20	0.4	6.3	24	29	65	2			2
59	25	G2A1	36		160	110	2	100	1	28	0.6	7.2	76	78	165	2	2	+	LSCS	150	100	1	100	1.3	26	0.6	6.5	45	50	107	2	1	+	140	90	1	150	1.6	25	0.6	5.9	40	40	99	2	1		2
60	27	Primi	37		160	100	1	100	1.6	18	0.5	6.8	29	23	71	1	1		LSCS	160	90	1	100	1.6	18	0.5	6.7	29	23	70	2	1		150	80	trace	100	1.6	18	0.4	6.3	29	23	70	2			2
61	26	G3P2L2	38		160	110	1	100	1.2	24	0.7	7.6	32	38	96	2	1		NVD	160	100	1	100	1.2	24	0.7	6.9	32	38	95	2	1		140	90	trace	150	1.3	22	0.6	5.1	32	38	95	2			2
62	25	G2P1L1	40		150	100	2	50	1.3	26	0.6	6.3	26	25	85	2	1		NVD	140	90	1	100	1.3	25	0.6	5.9	26	25	85	1			130	90	1	100	1.3	25	0.6	4.2	26	25	85	2			2
63	24	Primi	39		160	120	2	100	1.1	25	0.6	6	28	27	98	2	2	+	LSCS	150	100	1	150	1.1	25	0.5	5.1	28	27	96	2	1	+	140	90	trace	150	1.2	25	0.5	4.7	28	27	95	2			2
64	20	Primi	32	**headache,decreased urine output,**	170	110	3	30	1	35	0.9	8.8	72	71	183	2	2	+	NVD	150	100	2	50	1.3	28	0.7	7.9	55	51	155	2	1	+	140	90	1	100	1.5	26	0.7	6	43	39	82	2			2
65	24	G2A1	40		150	110	2	100	1.5	21	0.5	6.2	31	39	73	2	1		LSCS	140	90	1	100	1.4	20	0.5	5.5	31	35	70	1			130	80	trace	100	1.2	20	0.9	5	31	35	70	2			2
66	23	Primi	37		160	110	1	50	1.4	23	0.3	6.3	29	29	74	2	1		LSCS	150	100	1	100	1.4	22	0.3	5.1	29	28	74	2	1		140	90	trace	100	1.5	21	0.3	4.8	29	28	74	2			2
67	19	Primi	34		150	110	1	100	1.6	18	0.7	5.9	37	35	95	2	1		NVD	140	100	trace	100	1.6	18	0.6	5	37	34	94	2	1		140	90	trace	100	1.7	18	0.6	4.5	37	34	94	2			2
68	20	G2A1	38		160	110	1	150	1.5	20	0.6	6.8	30	38	93	1	1		LSCS	150	100	1	150	1.5	20	0.5	6.5	30	36	93	2	1		140	90	1	200	1.6	19	0.5	4.9	30	36	93	2			2
69	21	G3A2	37		160	100	2	150	1.4	20	0.6	6.5	30	34	96	1	1		LSCS	160	90	1	150	1.4	20	0.6	6	30	34	95	2	1		140	90	trace	150	1.5	20	0.5	5	30	32	92	2			2
70	24	G2P1L1	37	**epigastric pain, headache**	150	110	2	100	1.8	18	0.5	5.9	35	39	71	1	1	+	NVD	140	100	1	100	1.9	18	0.5	5	34	36	69	2	1	+	130	90	trace	150	1.9	18	0.5	4.5	34	36	94	2			2
71	18	Primi	36	**epigastric pain ,decreased urine output**	160	120	3	30	1	36	0.9	8.9	101	107	206	2	1	+	LSCS	150	100	2	150	1.5	30	0.7	7.2	88	86	154	2	1	+	140	90	1	100	1.6	26	0.7	6.1	46	40	96	2			2
72	27	G2P1L0	38		170	100	2	100	1.2	22	0.4	5.8	28	25	78	2	1	+	LSCS	150	90	1	100	1.2	21	0.4	5	28	25	76	2	1	+	140	90	1	100	1.4	21	0.3	4.8	28	25	76	2			2
73	25	G2P1L2	37		160	100	2	100	1.3	24	0.7	5.7	37	39	76	2	1		NVD	150	90	1	150	1.4	23	0.6	4.9	35	37	75	2	1		140	90	trace	150	1.4	22	0.6	4.3	35	37	75	2			2
74	28	Primi	36		160	110	1	50	1.5	17	0.6	6.8	95	98	164	2	1	+	LSCS	150	100	trace	100	1.5	17	0.5	6.5	72	78	111	2	1	+	140	90	trace	100	1.5	16	0.5	5.9	50	49	93	2			2
75	31	Primi	35		150	110	2	100	1.9	16	0.3	6.5	24	25	66	2	1		NVD	150	100	1	100	1.9	15	0.3	6	24	24	65	2	1		140	90	trace	150	1.4	15	0.2	5.3	24	24	65	2			2
76	32	G4P3L3	37		160	100	2	100	1.8	16	0.4	6.4	37	39	72	2	1		NVD	140	100	1	150	1.8	16	0.4	5.9	35	37	72	2	1		130	90	trace	150	2	15	0.4	4	35	37	72	2			2
77	21	Primi	38	**headache ,decreased urine output**	170	100	2	40	1	35	0.8	8.7	39	40	86	2	1	+	LSCS	160	90	1	100	1.3	30	0.7	7.9	38	40	80	2	1	+	140	90	1	100	1.5	26	0.7	6.1	35	39	80	2			2
78	23	G2P1L0	34	**headache ,decreased urine output**	160	110	2	50	1	33	0.8	8.5	61	59	164	2	1	+	LSCS	150	90	1	100	1.2	29	0.6	7.8	56	56	103	2	1	+	140	90	1	100	1.4	25	0.6	6	42	40	80	2			2
79	30	Primi	38		160	100	1	100	1.7	20	0.7	6.6	31	33	74	2	1		NVD	160	90	trace	150	1.7	20	0.6	6.6	31	32	72	2	1		140	90	trace	150	1.7	20	0.5	5.3	31	30	70	2			2
80	28	G3P1L1A1	37		160	110	1	100	1.6	21	0.7	6.5	22	26	66	2	1		NVD	150	100	trace	100	1.6	21	0.6	5.5	22	26	66	2	1		140	90	trace	150	1.6	19	0.6	4.8	22	25	64	2			2
81	31	G3P1L1A1	36		160	110	1	100	1	24	0.8	7.8	32	36	97	2	2		NVD	150	100	1	100	1.5	24	0.8	6.2	32	36	96	2	2		140	90	trace	150	1.5	23	0.7	5.9	32	36	95	2			2
82	29	G2A1	36		160	110	2	50	1.2	22	0.7	7.2	34	30	95	2	2		LSCS	150	90	1	100	1.2	21	0.7	6	32	30	95	2	1		140	90	1	100	1.3	20	0.7	4.8	32	30	95	2			2

S.No	Age	Parity	Gestational age(wks)	CASES - CURETTAGE DONE PATIENTS																DAY 1														DAY 2														DURATION OF STAY IN CCU/LA
				Imminent symptoms	systolic BP(mmHg)	diastolic BP(mmHg)	Urine albumin	Urine output	Platelet count (lak/hs/litre	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	Labetalol	Nifidepine	MgSO4	Mode of termination		systolic BP mm//Hg	diastolicBP mm/Hg	Urine albumin	Urine output (ml/hr)	Platelet count lak/lit	Bl.Ureamg/dl	S.Creatinineng/dl	Uric acidmg/dl	SGOTmeq/lt	SGPTmeq/lt	SAPmeq/lt	Labetalol	Nifidepine	MgSO4		systolicBPmm/Hg	diastolicBPmm/Hg	Urine albumin	Urine output(ml/hr)	Platelet countlakh	Bl.Ureamg/dl	S.Creatinineng/dl	Uric acidmg/dl	SGOTmeq/li	SGPTmeq/lt	SAPmeq/lt	Labetalol	Nifidepine	MgSO4	
83	27	Primi	37		150	100	2	100	1.4	26	0.6	6.3	28	25	87	2	2		LSCS		140	90	1	150	1.5	26	0.6	4.2	28	25	86	2				130	90	trace	150	1.5	25	0.6	4	28	25	86	2			1
84	22	Primi	30	**decreased urine output,**	170	120	3	30	1	30	0.9	9.2	72	78	186	2	2	+	NVD		150	100	2	50	1.4	28	0.8	5.8	69	63	150	2	2	+		140	90	1	100	1.7	23	0.7	4.1	42	40	97	2	1		2
85	24	G2A1	36	**headache, epigastric pain**	160	120	2	50	1.2	23	0.8	6.8	99	99	192	1	1	+	NVD		160	90	trace	100	2	23	0.8	5	83	78	137	2	1	+		130	90	trace	150	2.1	22	0.6	4	45	48	94	2			2
86	30	G2P1L0	37		150	110	2	100	1.8	18	0.5	6	43	35	75	1	1		LSCS		140	100	1	100	1.8	18	0.5	4.9	43	35	74	2	1			130	90	trace	100	1.8	16	0.4	4.7	42	35	74	2			2
87	32	G3P2L2	37		150	100	3	150	1.5	18	0.5	5.9	34	38	66	2	2		NVD		140	90	2	150	1.5	18	0.5	5.8	33	37	90	2				130	80	1	200	1.6	18	0.4	4.9	33	37	90	1			2
88	29	Primi	37		170	110	2	100	1.6	26	0.6	6.8	39	37	91	2	2	+	LSCS		150	100	1	150	1.6	25	0.6	6.6	39	37	90	2	2	+		140	90	trace	150	1.7	26	0.6	5	39	37	90	2			2
89	27	Primi	38		160	100	1	100	1.5	18	0.5	7.2	31	35	91	2	2		LSCS		150	100	1	100	1.5	18	0.5	7.1	30	35	91	2	2			150	90	1	150	1.5	16	0.5	6.1	30	35	91	2	1		2
90	29	G2A1	37		150	110	2	100	1.8	20	0.5	7.9	32	34	82	2	2		NVD		140	100	1	100	1.8	20	0.5	6.8	32	34	80	2	1			140	90	1	100	2	18	0.5	5.9	32	34	80	2			2
91	30	Primi	37		150	100	2	50	1.4	22	0.4	7	49	45	98	2	2		NVD		140	90	2	100	1.6	22	0.3	6	44	43	95	2				130	90	1	150	1.6	21	0.4	5.1	42	42	94	2			1
92	31	Primi	38		150	100	2	50	1.5	24	0.6	6.8	36	38	87	2	2		NVD		140	100	2	100	1.5	24	0.5	5.9	34	36	87	2	1			140	90	1	100	1.5	24	0.5	5.7	34	35	86	2			2
93	30	G3P1L1D1	38		160	100	2	100	1.7	26	0.5	6.4	34	39	89	2	2		NVD		140	90	1	150	1.6	25	0.5	6	34	36	88	2				130	90	trace	150	1.7	25	0.5	5.4	32	34	87	2			2
94	29	G3P2L2	38		160	100	2	100	1.9	28	0.7	6.2	22	28	89	2	2		LSCS		150	100	1	100	1.9	28	0.7	5.8	22	27	87	2	2			140	90	trace	100	2	28	0.7	5	22	27	87	2	1		2
95	26	Primi	37		160	100	2	100	1.6	20	0.6	6.6	35	30	88	2	2		NVD		150	100	2	100	1.6	20	0.6	6	35	29	87	2	2			130	90	1	150	1.6	18	0.5	5.1	35	28	86	2	1		2
96	27	Primi	40		150	100	2	100	1.5	18	0.5	6.1	34	31	96	2	2		LSCS		140	100	1	150	1.5	18	0.5	5.4	34	31	90	2	1			130	90	1	150	1.6	17	0.5	5	34	31	90	2			2
97	25	G3A2	32	**decreased urine output, headahe**	150	110	3	30	1	32	0.9	8.8	72	69	158	1	1	+	NVD		140	100	2	100	1.3	26	0.8	6.6	43	40	98	2	1	+		130	90	1	150	1.5	25	0.6	5.3	40	39	96	2			2
98	25	G2A1	36		160	110	1	100	1.5	28	0.5	7	36	31	85	1	1		NVD		150	90	1	100	1.5	24	0.5	6.2	35	31	80	2	1			140	90	trace	150	1.6	22	0.4	5.9	34	30	80	2			2
99	23	Primi	37		150	100	2	50	1.4	26	0.4	6.8	38	32	97	1	1		NVD		140	100	1	100	1.3	24	0.3	6.1	35	32	95	2	1			140	90	trace	100	1.5	20	0.3	5.8	35	32	94	2			2
100	28	G3P1L1	37		160	100	1	100	1.3	25	0.6	6.1	39	35	87	2	2		LSCS		150	90	trace	150	1.4	25	0.5	5.7	37	34	84	2				140	80	trace	150	1.4	25	0.5	5.7	37	34	84	2			2

S.No	Age	Parity	Gestational age(wks)	CONTROLS - CURETTAGE NOT DONE																DAY 1																				DAY 2																Duration of stay in hospital
				Imminent symptoms	systolic BP(mm/Hg)	diastolic BP(mm/Hg)	urine albumin	Urine output(ml/hr)	Platelet count (lakhs/litre)	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	labetalol	Nifidepine	MgSO4	Mode of termination		systolic BP(mm/Hg)	diastolicBP(mmHg)	urine albumin	Urine output(ml/hr)	Platelet count (lakhs/lit	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	labetalol	Nifidepine	MgSO4		systolicBP(mmHg)	diastolicBP(mmHg)	urine albumin	Urine output(ml/hr)	Platelet count (lakhs/lit	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	labetalol	Nifidepine	MgSO4	
1	30	G2P1L1	37		160	110	2	100	1.5	22	0.6	6.3	34	36	76	2	2		LSCS		160	100	2	100	1.5	22	0.6	6.3	34	36	76	2	2		150	100	1	100	1.6	21	0.6	6	34	36	76	2	2		3
2	25	G2P1L0	38		160	110	1	100	1.6	25	0.7	6.5	42	40	97	2	2		NVD		150	110	1	100	1.5	25	0.7	6.3	40	40	96	2	2		150	100	trace	100	1.7	25	0.6	6	40	39	95	2	2		3
3	18	Primi	34	headache ,decreased urine output	170	120	2	30	1	38	0.9	8.5	75	79	154	2	2	+	NVD		160	110	2	50	1.1	38	0.8	8	62	69	111	2	2	+	160	100	1	50	1.1	37	0.8	7.9	62	68	110	2	2	+	3
4	20	Primi	37		160	110	2	100	1.5	26	0.7	6.4	38	36	87	2	2		LSCS		150	100	1	100	1.5	25	0.7	6.3	38	36	87	2	2		150	90	1	100	1.5	25	0.7	6.2	38	36	86	2	1		2
5	31	G3P1L1A1	37		150	110	1	100	1.7	21	0.7	5.9	32	34	79	2	2		LSCS		150	110	1	50	1.7	21	0.7	5.8	32	34	79	2	2		140	100	trace	50	1.8	21	0.7	5.8	31	33	78	2	2		2
6	21	Primi	34		150	100	1	50	1.3	29	0.6	7.2	25	27	66	2	2		NVD		160	100	1	100	1.3	29	0.6	7	24	25	66	2	2		150	100	1	100	1.3	27	0.6	7	23	25	65	2	2		3
7	22	Primi	36		160	110	2	50	1.8	26	0.5	7	42	39	87	2	2		LSCS		150	100	2	50	1.8	26	0.5	7	42	38	87	2	2		150	100	2	100	1.8	26	0.4	6.9	41	38	86	2	2		3
8	19	Primi	34	headache	160	110	2	50	1.3	18	0.5	7.1	36	38	85	2	2	+	NVD		160	100	1	100	1.3	18	0.5	7	36	38	85	2	2	+	150	100	1	100	1.4	18	0.5	7	36	38	85	2	2	+	3
9	28	G2A1	37		160	90	1	100	1.9	16	0.3	6.9	37	35	79	2	2		LSCS		150	90	1	100	1.9	16	0.3	6.8	37	35	79	2	1		140	90	1	100	1.9	16	0.3	6.5	37	35	79	2			2
10	20	Primi	37		150	110	1	100	1.6	20	0.4	6.8	30	32	88	2	2		LSCS		140	100	1	100	1.6	20	0.4	6.5	30	32	87	2	2		140	100	trace	100	1.7	20	0.4	6.5	30	32	87	2	2		2
11	18	Primi	38		160	100	1	100	1.5	21	0.6	6.7	31	32	91	2	2		NVD		150	100	1	150	1.5	21	0.6	6.6	31	32	90	2	2		150	100	1	100	1.5	20	0.6	6.6	31	32	91	2	2		3
12	24	Primi	39		160	110	2	100	1.3	22	0.6	6	39	40	96	2	2		NVD		160	100	2	150	1.3	22	0.6	6	39	40	96	2	2		150	100	1	150	1.4	21	0.5	5.9	38	40	95	2	2		3
13	22	Primi	40		160	100	1	150	1.2	19	0.5	6.7	41	40	98	2	2		LSCS		160	90	1	100	1.4	19	0.5	6.7	40	40	98	2	1		150	90	trace	100	1.4	19	0.5	6.6	40	39	95	2	1		2
14	20	G2A1	30	headache ,decreased urine output	160	110	2	100	1	37	0.8	9	72	78	169	1	1	+	LSCS		160	100	2	50	1	35	0.7	8.5	69	67	119	2	2	+	160	100	2	50	1	35	0.7	8	65	66	110	2	2	+	3
15	21	Primi	38		150	110	2	30	1.4	25	0.7	7.2	41	38	94	2	2		LSCS		150	100	1	100	1.4	25	0.7	7.1	40	38	93	2	2		150	90	1	100	1.4	23	0.6	6.9	40	38	92	2	1		2
16	29	G2P1L1	37		160	100	2	100	1.6	24	0.6	5.9	32	30	88	2	2		NVD		150	90	1	100	1.5	24	0.6	5.7	32	30	88	2	1		140	90	1	100	1.5	24	0.6	5.5	32	30	88	2			2
17	20	G2A1	37		150	100	2	100	1.7	25	0.7	6	35	35	97	2	2		NVD		140	100	2	100	1.7	24	0.7	6	35	35	97	2	2		140	90	1	100	1.8	23	0.7	5.7	35	35	97	2			2
18	19	Primi	34	headache , blurring of vision	160	120	2	100	1	39	0.8	8.9	75	79	151	2	2	+	NVD		160	110	2	50	1	37	0.8	8.5	74	73	132	2	2	+	150	100	1	50	1.1	37	0.8	8.4	73	71	112	2	2	+	3
19	26	G2P1L1	37		160	100	2	50	1.8	24	0.3	6.8	34	32	85	1	1		LSCS		150	100	1	50	1.8	24	0.3	6.5	34	32	85	2	2		150	90	1	100	1.9	22	0.3	6.5	32	32	84	2	1		2
20	23	Primi	37		160	100	1	50	1.6	23	0.5	5.9	38	36	80	2	2		LSCS		150	90	trace	100	1.6	23	0.5	5.7	38	35	84	2	1		150	90	trace	100	1.7	23	0.5	5.7	38	34	84	2	1		2
21	28	Primi	37		170	120	1	100	1.5	15	0.6	5.8	37	37	98	2	2	+	NVD		160	100	1	150	1.5	15	0.5	5.8	37	36	97	2	2	+	150	100	1	150	1.6	15	0.5	5.5	37	36	96	2	2	+	3
22	23	Primi	37		150	100	2	150	1.4	16	0.7	6.1	30	39	91	1	1		LSCS		140	100	1	100	1.5	16	0.7	6	30	35	91	2	2		140	90	1	100	1.5	15	0.7	6	30	34	90	2			2
23	22	Primi	40		160	110	2	100	1.3	20	0.3	5.7	32	31	95	2	2		LSCS		150	100	2	100	1.4	20	0.3	5.5	32	31	95	2	2		150	90	2	100	1.4	20	0.3	5.5	32	31	95	2	1		2
24	21	Primi	38	decreased urine output	170	110	3	50	1.1	33	0.9	8.8	85	89	175	2	2	+	NVD		160	100	2	50	1.2	33	0.8	8	85	85	154	2	2	+	160	100	2	50	1.1	32	0.8	8	85	82	118	2	2	+	3
25	27	G3P1L1D1	39		150	110	1	30	1.6	22	0.5	6.9	38	35	96	2	2		NVD		140	100	trace	150	1.5	22	0.5	6.5	38	35	96	2	2		140	90	trace	150	1.6	21	0.5	6.3	38	34	95	2			2
26	19	G2A1	38		160	100	1	150	1.5	21	0.6	6.6	38	37	92	2	2		LSCS		150	100	1	100	1.8	21	0.6	6.6	38	37	92	2	2		140	100	1	100	1.8	21	0.5	6.5	37	36	92	2	2		2
27	25	G2P1LO	37		160	100	2	100	1.7	22	0.6	6.5	39	38	79	2	2		NVD		150	90	1	100	1.7	22	0.5	6.5	39	37	78	2	1		150	90	trace	100	1.8	22	0.5	6.5	39	37	78	2	1		2
28	20	Primi	37	headache , blurring of vision	160	120	2	50	1.1	29	0.8	8.1	79	76	121	2	2	+	NVD		160	110	2	50	1.1	29	0.8	8	78	76	119	2	2	+	150	110	2	50	1.2	27	0.7	7.5	78	75	117	2	2	+	4
29	22	Primi	36		150	100	1	150	1.5	24	0.7	7.2	32	30	68	2	2		NVD		150	90	1	150	1.5	24	0.7	7	32	30	68	2	1		140	90	1	150	1.6	24	0.7	7	32	30	65	2			2
30	23	Primi	37		150	110	1	100	1.4	26	0.5	7.3	41	40	93	2	2		LSCS		150	100	1	100	1.4	26	0.5	7.3	41	40	92	2	2		140	100	trace	150	1.4	25	0.5	7.2	41	40	91	2	2		2
31	29	G2P1L1	36		160	100	2	100	1.3	25	0.6	6.8	39	30	75	2	2		LSCS		150	90	1	100	1.3	25	0.6	6.5	39	30	75	2	1		150	90	1	100	1.4	25	0.6	6.5	39	30	74	2	1		2
32	31	G2P1L1	37		160	110	1	50	1.2	24	0.6	6.7	37	36	99	2	2		LSCS		150	100	1	50	1.2	24	0.6	6.5	37	36	99	2	2		150	90	1	50	1.3	24	0.6	6.4	37	36	97	2	1		3
33	22	G2A1	37		150	110	1	100	1.6	17	0.5	6	45	40	97	2	2		LSCS		140	100	trace	100	1.6	17	0.5	6	45	40	97	2	2		140	100	trace	100	1.6	17	0.5	6	45	40	96	2	2		3
34	26	Primi	34	headache	170	110	1	50	1.5	18	0.4	6	30	30	88	2	2	+	NVD		160	100	1	50	1.5	18	0.4	5.8	29	29	88	2	2	+	150	90	1	150	1.5	18	0.4	5.8	29	28	90	2	1	+	2
35	25	Primi	36		160	100	2	100	1.1	16	0.3	6.1	31	33	73	2	2		LSCS		150	100	1	100	1.1	16	0.3	6	31	33	73	2	2		150	90	trace	100	1.1	16	0.3	6	31	32	73	2	1		2
36	20	Primi	37		160	110	2	100	1.3	20	0.7	6.3	34	32	82	2	2		LSCS		160	100	2	100	1.3	20	0.6	6	34	32	81	2	2		150	90	1	100	1.3	20	0.8	5.9	34	31	80	2	1		2
37	32	Primi	37		160	110	1	100	1.5	22	0.6	6.3	36	38	89	2	2		NVD		160	100	2	100	1.5	22	0.6	6.3	35	37	90	2	2		150	100	1	100	1.6	21	0.6	6	35	37	90	2	2		4
38	21	Primi	36		150	100	2	100	1.6	25	0.7	6.5	38	38	90	2	2		NVD		150	110	1	100	1.5	25	0.7	6.3	38	38	90	2	2		150	100	trace	100	1.7	25	0.6	6	38	38	90	2	2		4
39	18	Primi	34	decreased urine output	170	120	2	30	1	38	0.9	8.5	79	81	152	2	2	+	NVD		160	110	2	50	1.1	38	0.8	8	78	79	143	2	2	+	160	100	1	50	1.1	37	0.8	7.9	77	77	126	2	2	+	4
40	27	G2P1L2	37		160	110	1	100	1.5	26	0.7	6.4	48	46	85	2	2		LSCS		160	100	1	100	1.5	25	0.7	6.3	43	46	85	2	2		150	90	1	100	1.5	25	0.7	6.2	43	46	85	2	1		2
41	24	Primi	37		150	100	1	50	1.7	21	0.7	5.9	41	39	89	2	2		NVD		150	110	1	50	1.7	21	0.7	5.8	40	39	87	2	2		140	100	trace	50	1.8	21	0.7	5.8	40	39	86	2	2		3
42	23	G2A1	38		170	100	2	50	1.3	19	0.6	7.2	38	33	79	2	2		NVD		160	100	1	100	1.3	29	0.6	7	38	33	78	2	2		150	100	1	100	1.3	27	0.6	7	38	33	78	2	2		3
43	25	G2P1L1	38		160	100	2	50	1.8	26	0.5	7	34	36	86	2	2		NVD		150	100	2	50	1.8	26	0.5	7	34	36	86	2	2		150	100	2	100	1.8	26	0.4	6.9	34	35	85	2	2		3
44	22	Primi	39		170	110	1	100	1.3	18	0.5	7.1	36	38	91	2	2		NVD		160	100	1	100	1.3	18	0.5	7	36	38	90	2	2		150	100	1	100	1.4	18	0.5	7	36	37	90	2	2		3
45	26	Primi	38		160	90	1	100	1.9	16	0.3	6.9	37	35	90	2	2		LSCS		150	90	1	100	1.9	16	0.3	6.8	37	35	90	2	1		140	90	1	100	1.9	16	0.3	6.5	37	34	89	2			2
46	24	G2A1	37		150	110	1	100	1.6	20	0.4	6.8	30	32	88	2	2		LSCS		140	100	1	100	1.6	20	0.4	6.5	30	32	88	2	2		140	100	trace	100	1.7	20	0.4	6.5	30	32	88	2	2		3
47	23	Primi	37		160	100	2	100	1.5	21	0.6	6.7	31	32	90	2	2		LSCS		150	100	1	100	1.5	21	0.6	6.6	31	32	90	2	2		150	100	1	100	1.5	20	0.6	6.6	31	31	89	2	2		3
48	21	Primi	36		160	110	2	150	1.3	22	0.6	6	39	35	93	2	2		NVD		160	100	2	150	1.3	22	0.6	6	39	35	92	2	2		150	100	1	150	1.4	21	0.5	5.9	39	35	91	2	2		3
49	22	G3A2	34	headache ,decreased urine output	160	110	2	30	1	37	0.8	8.9	87	89	153	2	2	+	NVD		160	100	2	50	1	35	0.7	8.5	85	84	123	2	2	+	160	100	2	50	1	35	0.7	8	84	84	112	2	2	+	4
50	24	Primi	37		150	110	2	100	1.4	25	0.7	7.2	36	37	89	2	2		LSCS		150	100	1	100	1.4	25	0.7	7.1	35	36	88	2	2		150	90	1	100	1.4	23	0.6	6.9	35	34	88	2	1		2

S.No	Age	Parity	Gestational age(wks)	CONTROLS - CURETTAGE NOT DONE																DAY 1																		DAY 2															Duration of stay in hospital
				Imminent symptoms	systolic BP(mmHg)	diastolic BP(mmHg)	urine albumin	Urine output(ml/hr)	Platelet count (lakhs/litre)	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	labetalol	Nifidepine	MgSO4	Mode of termination	systolic BP(mmHg)	diastolicBP(mmHg)	urine albumin	Urine output(ml/hr)	Platelet count (lakhs/litre)	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	labetalol	Nifidepine	MgSO4	systolicBP(mmHg)	diastolicBP(mmHg)	urine albumin	Urine output(ml/hr)	Platelet count (lakhs/litre)	Bl.Urea(mg/dl)	S.Creatinine(mg/dl)	Uric acid(mg/dl)	SGOT(mEq/L)	SGPT(mEq/L)	SAP(mEq/L)	labetalol	Nifidepine	MgSO4	
51	28	G3P2L2	30	headache ,decreased urine output	160	110	2	100	1	37	0.8	9	72	78	169	1	1	+	NVD	160	100	2	50	1	35	0.7	8.5	69	67	119	2	2	+	160	100	2	50	1	35	0.7	8	65	66	110	2	2	+	3
52	32	G2P1L1	38		150	110	2	30	1.4	25	0.7	7.2	41	38	94	2	2		LSCS	150	100	1	100	1.4	25	0.7	7.1	40	38	93	2	2		150	90	1	100	1.4	23	0.6	6.9	40	38	92	2	1		2
53	27	G2P1L1	37		160	100	2	100	1.6	24	0.6	5.9	32	30	88	2	2		NVD	150	90	1	100	1.5	24	0.6	5.7	32	30	88	2	1		140	90	1	100	1.5	24	0.6	5.5	32	30	88	2			2
54	24	Primi	37		150	100	2	100	1.7	25	0.7	6	35	35	97	2	2		NVD	140	100	2	100	1.7	24	0.7	6	35	35	97	2	2		140	90	1	100	1.8	23	0.7	5.7	35	35	97	2			2
55	24	G2A1	34	headache , blurring of vision	160	120	2	100	1	39	0.8	8.9	75	79	151	2	2	+	NVD	160	110	2	50	1	37	0.8	8.5	74	73	132	2	2	+	150	100	1	50	1.1	37	0.8	8.4	73	71	112	2	2	+	3
56	27	G2P1L0	37		160	100	2	50	1.8	24	0.3	6.8	34	32	85	1	1		LSCS	150	100	1	50	1.8	24	0.3	6.5	34	32	85	2	2		150	90	1	100	1.9	22	0.3	6.5	32	32	84	2	1		2
57	19	Primi	37		160	100	1	50	1.6	23	0.5	5.9	38	36	80	2	2		LSCS	150	90	trace	100	1.6	23	0.5	5.7	38	35	84	2	1		150	90	trace	100	1.7	23	0.5	5.7	38	34	84	2	1		2
58	27	Primi	37		170	120	1	100	1.5	15	0.6	5.8	37	37	98	2	2	+	NVD	160	100	1	150	1.5	15	0.5	5.8	37	36	97	2	2	+	150	100	1	150	1.6	15	0.5	5.5	37	36	96	2	2	+	3
59	22	Primi	37		150	100	2	150	1.4	16	0.7	6.1	30	39	91	1	1		LSCS	140	100	1	100	1.5	16	0.7	6	30	35	91	2	2		140	90	1	100	1.5	15	0.7	6	30	34	90	2			2
60	29	G2P1L0	40		160	110	2	100	1.3	20	0.3	5.7	32	31	95	2	2		LSCS	150	100	2	100	1.4	20	0.3	5.5	32	31	95	2	2		150	90	2	100	1.4	20	0.3	5.5	32	31	95	2	1		2
61	19	Primi	38	decreased urine output	170	110	3	50	1.1	33	0.9	8.8	85	89	175	2	2	+	LSCS	160	100	2	50	1.2	33	0.8	8	85	85	154	2	2	+	160	100	2	50	1.1	32	0.8	8	85	82	118	2	2	+	3
62	20	G2A1	39		150	110	1	30	1.6	22	0.5	6.9	38	35	96	2	2		LSCS	140	100	trace	150	1.5	22	0.5	6.5	38	35	96	2	2		140	90	trace	150	1.6	21	0.5	6.3	38	34	95	2			2
63	31	Primi	38		160	100	1	150	1.5	21	0.6	6.6	38	37	92	2	2		LSCS	150	100	1	100	1.8	21	0.6	6.6	38	37	92	2	2		140	100	1	100	1.8	21	0.5	6.5	37	36	92	2	2		2
64	30	G2P1L1	37		160	100	2	100	1.7	22	0.6	6.5	39	38	79	2	2		NVD	150	90	1	100	1.7	22	0.5	6.5	39	37	78	2	1		150	90	trace	100	1.8	22	0.5	6.5	39	37	78	2	1		2
65	21	Primi	37	headache , blurring of vision	160	120	2	50	1.1	29	0.8	8.1	79	76	121	2	2	+	LSCS	160	110	2	50	1.1	29	0.8	8	78	76	119	2	2	+	150	110	2	50	1.2	27	0.7	7.5	78	75	117	2	2	+	4
66	23	Primi	36		150	100	1	150	1.5	24	0.7	7.2	32	30	68	2	2		LSCS	150	90	1	150	1.5	24	0.7	7	32	30	68	2	1		140	90	1	150	1.6	24	0.7	7	32	30	65	2			2
67	28	Primi	37		150	110	1	100	1.4	26	0.5	7.3	41	40	93	2	2		LSCS	150	100	1	100	1.4	26	0.5	7.3	41	40	92	2	2		140	100	trace	150	1.4	25	0.5	7.2	41	40	91	2	2		2
68	24	Primi	36		160	100	2	100	1.3	25	0.6	6.8	39	30	75	2	2		LSCS	150	90	1	100	1.3	25	0.6	6.5	39	30	75	2	1		150	90	1	100	1.4	25	0.6	6.5	39	30	74	2	1		2
69	21	Primi	37		160	110	1	50	1.2	24	0.6	6.7	37	36	99	2	2		LSCS	150	100	1	50	1.2	24	0.6	6.5	37	36	99	2	2		150	90	1	50	1.3	24	0.6	6.4	37	36	97	2	1		3
70	29	G2P1L1	37		150	110	1	100	1.6	17	0.5	6	45	40	97	2	2		LSCS	140	100	trace	100	1.6	17	0.5	6	45	40	97	2	2		140	100	trace	100	1.6	17	0.5	6	45	40	96	2	2		3
71	24	G2P1L1	34	headache	170	110	1	50	1.5	18	0.4	6	30	30	88	2	2	+	NVD	160	100	1	50	1.5	18	0.4	5.8	29	29	88	2	2	+	150	90	1	150	1.5	18	0.4	5.8	29	28	90	2	1	+	2
72	25	Primi	36		160	100	2	100	1.1	16	0.3	6.1	31	33	73	2	2		LSCS	150	100	1	100	1.1	16	0.3	6	31	33	73	2	2		150	90	trace	100	1.1	16	0.3	6	31	32	73	2	1		2
73	30	Primi	37		160	110	2	100	1.3	20	0.7	6.3	34	32	82	2	2		LSCS	160	100	2	100	1.3	20	0.6	6	34	32	81	2	2		150	90	1	100	1.3	20	0.8	5.9	34	31	80	2	1		2
74	19	Primi	37		160	110	1	100	1.5	22	0.6	6.3	36	38	89	2	2		LSCS	160	100	2	100	1.5	22	0.6	6.3	35	37	90	2	2		150	100	1	100	1.6	21	0.6	6	35	37	90	2	2		4
75	26	G3A2	36		150	100	2	100	1.6	25	0.7	6.5	38	38	90	2	2		NVD	150	110	1	100	1.5	25	0.7	6.3	38	38	90	2	2		150	100	trace	100	1.7	25	0.6	6	38	38	90	2	2		4
76	28	G2A1	34	decreased urine output	170	120	2	30	1	38	0.9	8.5	79	81	152	2	2	+	NVD	160	110	2	50	1.1	38	0.8	8	78	79	143	2	2	+	160	100	1	50	1.1	37	0.8	7.9	77	77	126	2	2	+	4
77	22	Primi	37		160	110	1	100	1.5	26	0.7	6.4	48	46	85	2	2		LSCS	160	100	1	100	1.5	25	0.7	6.3	43	46	85	2	2		150	90	1	100	1.5	25	0.7	6.2	43	46	85	2	1		2
78	31	Primi	37		150	100	1	50	1.7	21	0.7	5.9	41	39	89	2	2		LSCS	150	110	1	50	1.7	21	0.7	5.8	40	39	87	2	2		140	100	trace	50	1.8	21	0.7	5.8	40	39	86	2	2		3
79	20	Primi	38		170	100	2	50	1.3	19	0.6	7.2	38	33	79	2	2		NVD	160	100	1	100	1.3	29	0.6	7	38	33	78	2	2		150	100	1	100	1.3	27	0.6	7	38	33	78	2	2		3
80	26	G2P1L1	38		160	100	2	50	1.8	26	0.5	7	34	36	86	2	2		LSCS	150	100	2	50	1.8	26	0.5	7	34	36	86	2	2		150	100	2	100	1.8	26	0.4	6.9	34	35	85	2	2		3
81	25	Primi	34	headache ,decreased urine output	170	120	2	30	1	38	0.9	8.5	75	79	154	2	2	+	NVD	160	110	2	50	1.1	38	0.8	8	62	69	111	2	2	+	160	100	1	50	1.1	37	0.8	7.9	62	68	110	2	2	+	3
82	22	G2A1	37		160	110	2	100	1.5	26	0.7	6.4	38	36	87	2	2		LSCS	160	100	1	100	1.5	25	0.7	6.3	38	36	87	2	2		150	90	1	100	1.5	25	0.7	6.2	38	36	86	2	1		2
83	27	G2P1L1	37		150	110	1	100	1.7	21	0.7	5.9	32	34	79	2	2		LSCS	150	110	1	50	1.7	21	0.7	5.8	32	34	79	2	2		140	100	trace	50	1.8	21	0.7	5.8	31	33	78	2	2		2
84	21	Primi	34		150	100	1	50	1.3	29	0.6	7.2	25	27	66	2	2		NVD	160	100	1	100	1.3	29	0.6	7	24	25	66	2	2		150	100	1	100	1.3	27	0.6	7	23	25	65	2	2		3
85	29	G4P1L1D2	36		160	110	2	50	1.8	26	0.5	7	42	39	87	2	2		LSCS	150	100	2	50	1.8	26	0.5	7	42	38	87	2	2		150	100	2	100	1.8	26	0.4	6.9	41	38	86	2	2		3
86	24	G3A2	38	headache	160	110	2	50	1.3	18	0.5	7.1	36	38	85	2	2	+	LSCS	160	100	1	100	1.3	18	0.5	7	36	38	85	2	2	+	150	100	1	100	1.4	18	0.5	7	36	38	85	2	2	+	3
87	27	Primi	37		160	90	1	100	1.9	16	0.3	6.9	37	35	79	2	2		LSCS	150	90	1	100	1.9	16	0.3	6.8	37	35	79	2	1		140	90	1	100	1.9	16	0.3	6.5	37	35	79	2			2
88	25	Primi	37		150	110	1	100	1.6	20	0.4	6.8	30	32	88	2	2		LSCS	140	100	1	100	1.6	20	0.4	6.5	30	32	87	2	2		140	100	trace	100	1.7	20	0.4	6.5	30	32	87	2	2		2
89	22	Primi	38		160	100	1	100	1.5	21	0.6	6.7	31	32	91	2	2		NVD	150	100	1	150	1.5	21	0.6	6.6	31	32	90	2	2		150	100	1	100	1.5	20	0.6	6.6	31	32	91	2	2		3
90	23	Primi	39		160	110	2	100	1.3	22	0.6	6	39	40	96	2	2		NVD	160	100	2	150	1.3	22	0.6	6	39	40	96	2	2		150	100	1	150	1.4	21	0.5	5.9	38	40	95	2	2		3
91	30	G2P1L1	40		160	100	1	150	1.2	19	0.5	6.7	41	40	98	2	2		LSCS	160	90	1	100	1.4	19	0.5	6.7	40	40	98	2	1		150	90	trace	100	1.4	19	0.5	6.6	40	39	95	2	1		2
92	29	G2P1L0	30	headache ,decreased urine output	160	110	2	100	1	37	0.8	9	72	78	169	1	1	+	LSCS	160	100	2	50	1	35	0.7	8.5	69	67	119	2	2	+	160	100	2	50	1	35	0.7	8	65	66	110	2	2	+	3
93	19	Primi	38		150	110	2	30	1.4	25	0.7	7.2	41	38	94	2	2		LSCS	150	100	1	100	1.4	25	0.7	7.1	40	38	93	2	2		150	90	1	100	1.4	23	0.6	6.9	40	38	92	2	1		2
94	25	Primi	37		160	100	2	100	1.6	24	0.6	5.9	32	30	88	2	2		LSCS	150	90	1	100	1.5	24	0.6	5.7	32	30	88	2	1		140	90	1	100	1.5	24	0.6	5.5	32	30	88	2			2
95	22	G2P1L1	37		150	100	2	100	1.7	25	0.7	6	35	35	97	2	2		LSCS	140	100	2	100	1.7	24	0.7	6	35	35	97	2	2		140	90	1	100	1.8	23	0.7	5.7	35	35	97	2			2
96	24	G2P1L1	34	headache , blurring of vision	160	120	2	100	1	39	0.8	8.9	75	79	151	2	2	+	NVD	160	110	2	50	1	37	0.8	8.5	74	73	132	2	2	+	150	100	1	50	1.1	37	0.8	8.4	73	71	112	2	2	+	3
97	26	G3P1L1A1	37		160	100	2	50	1.8	24	0.3	6.8	34	32	85	1	1		LSCS	150	100	1	50	1.8	24	0.3	6.5	34	32	85	2	2		150	90	1	100	1.9	22	0.3	6.5	32	32	84	2	1		2
98	22	Primi	37		160	100	1	50	1.6	23	0.5	5.9	38	36	80	2	2		LSCS	150	90	trace	100	1.6	23	0.5	5.7	38	35	84	2	1		150	90	trace	100	1.7	23	0.5	5.7	38	34	84	2	1		2
99	21	Primi	37		170	120	1	100	1.5	15	0.6	5.8	37	37	98	2	2	+	LSCS	160	100	1	150	1.5	15	0.5	5.8	37	36	97	2	2	+	150	100	1	150	1.6	15	0.5	5.5	37	36	96	2	2	+	3
100	28	G2P1L0	37		150	100	2	150	1.4	16	0.7	6.1	30	39	91	1	1		LSCS	140	100	1	100	1.5	16	0.7	6	30	35	91	2	2		140	90	1	100	1.5	15	0.7	6	30	34	90	2			2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INTRODUCTION
Hypertensive disorders during pregnancy continue to be a major cause of maternal and perinatal morbidity and mortality worldwide .In developing countries they are second only to anemia with approximately 3- 10 %of all pregnancies.
Pritchard et al observed "in order to effect a complete cure from preeclampsia the chorionic villi must be expelled or surgically removed .Redger et al and music et al postulated that the endothelial cells are affected by a cytotoxic factor produced by the trophoblastic cells which is responsible for the pathophysiology of preeclampsia1.
One such theory states that "the decidua and amniotic fluid contains a toxin called HYSTEROTONIN which acts as a pressor substance leading to the development of preeclampsia.It is found in women with preeclampsia resolution of preeclampsia and eclampsia occurs by delivery and complete removal of trophoblastic cells.
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